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TRANSCRIPT OF PODCAST
Shouldn't call it a pandemic. What we were talking about is a, mass casualty event because of biowarfare programs. There's I've managed to acquire the, unusual clots that have been, banded around on the Internet. And, unfortunately, for, these clots were received by myself from Richard Hirschman's r. Now, before we sort of get lost in details, what I can tell you is is that this proteinaceous form is, like I said, highly it's misfolded, amyloidogenic fibrin.

You're dealing with a any systemic amyloid type disorder is considered along the sort of scale of, disorders that you you can turn up to a hospital with as being one of the more serious conditions that you can develop. Right? And people who have light chain amyloidosis, right, do not live as long. There's very little in the way of treatments. And as they progress through their illness, the reason that they die earlier is that the light chain amyloids penetrate into all their organs such that the organs shut down because they become gummed up with this, amyloidogenic peptide.

K? Mhmm. Now if we kind of explains a number of things that people might be seeing out there in the world. Yes. So this is this is something that I would encourage people to maintain, which is that because of the nature of this type of disorder, it'll it can manifest in a number of different ways.

Okay? Cancer is one. Neurodegenerative is another. In this instance, if your circulatory system is filling up with this undigestible amyloidogenic peptide, you're gonna develop all manner of cardiovascular and thrombotic type tissues or disease. And, again, I you know, if there's no desire on behalf of the authorities to look at this, because it essentially, the blowback would go to them anyway.

The buck stops with them in terms of how the public thinks. Right? And if people have lost loved ones, either due to exposure to this synthetic virus or due to a new just to, force public health measures using novel techniques that have never been deployed on mass before, where you where you've made the body express this toxic peptide that can act as a seed, right, to catalyze something into this well, I should be wearing gloves. Where you're getting grams of material post exposure. That's pretty dull.

It's very dire. That looks like there's an infective peptide if this sample that I've been running has been out of the body for three months. Even though it's been out of the body three months, once it comes into contact with fresh plasma, it's able to initiate the, the misfolding and aggregation into a amyloidogenic form such that it such that you're seeing an increase in

fluorescence. This is a big concern. So first off, I'm fairly confident that most blood products post COVID are likely contaminated with probably seed amounts of misfolded fib Hello, everyone.

Welcome back to Health Alliance Australia's podcast. My name is Jeanne Rose Anjwatha, and I'm here with neuroscientist and distinguished researcher, doctor McKeven McCann. Today, we are doing an important follow-up to our podcast recorded last year on bio warfare prions. Doctor McCann and other researchers have now confirmed through laboratory analysis that we have a global amelogenic health crisis. These amelogenic prion triggering epitopes and peptides are also seeded in the environment as well as in living organisms.

They're highly resistant with the possess potential to cause multiorgan dysfunction, neurodegeneration, cancer, heart disease, sudden death, strokes, etcetera, etcetera. Do watch our previous interview if you haven't already, which is linked in the description, an excellent utility video on prions c 19, biowarfare, historical context of prion diseases, animal models, etcetera. Doctor McCann has found a very novel kill mechanism. This research is cutting edge. The latest laboratory findings should be the biggest news story out there right now.

It affects every single person in the world. Due to censorship, the bulk of the podcast will be on Rumble, Bitchute, and Vimeo. Watch to the end. We will be discussing the section of amyloid burden through blood samples that you can send to doctor McCart's lab from anywhere in the world if you have any concerns or would or would like to simply submit a sample for research purposes. There will also be a link if you want to contribute financially to this important work, which is completely community funded.

Welcome, doctor McCann. Thank you for coming today. For those who don't know you, could you please do a short introduction, and then we can get right into the clinical findings and implications? Sure. So I think you did a pretty good job of sort of the more recent, but my history as it goes back into past decades was, as you're saying, neuroscience, systems neuroscience, disorders of corticobasal ganglia systems.

I developed the first primate model of Tourette Syndrome, but I also worked on, Parkinson's neurodegenerative disorders, surgical interventions thereof, around a technique called deep brain stimulation. And, basically, I got dragged into COVID very early on. I had a a very severe case from what was basically Asia's biggest super spreading event that came out of Korea. It was touch and go with me, as it sort of took a hold. And as that was sort of unfold, I was going through that, word began to come out of events going down in Wuhan.

And, you know, I'm familiar with most viruses just having, you know, you run into norovirus, influenzas, etcetera as you, traverse through this life. And this was something standout that, I had I ran into and as I was watching everything, unfurl. And I spoke up very early about

because I, you know, I had predisposing factors anyway. I was sort of semi medically retired because of a TBI, but, my interaction with COVID was what would be defined now as neuro COVID. It was, yeah.

It was just a tough, week few weeks, and then there was a very long recovery process afterwards. And I as people were talking about possibility of, leaks from Wuhan, I spoke up publicly and said, you people, you wanna pay attention to what this is. It's, you know, it did a a number on me, and it's obviously done a number on many other people. The, the issue you have is that, well, since we spoke last year, there's been a sort of sea change in how, the public not the public, the the institutes have, accepted the fact that there is a synthetic origin to COVID. The, what was very obviously a organized program, campaign within the, you know, initially, the people involved, whether that's like the EcoHealth Alliance, etcetera, just trying to sort of disseminate and sort of flood the scientific literature with, with basically scam articles as to what what it was we were dealing with, that it just it was just a bat virus.

It just it's just pure chance that it happened to arise next to China's premier center for study into coronaviruses, etcetera. And in the last week or two, we've had them admit that, members of the armed forces, for The US came back sick from the Wuhan games, military games in Wuhan, which literally was running whilst they ran event two zero one. And, you know, if if people wanna dig into those tabletop exercises, I would say what you've what or what we're going through right now is essentially a, it's it's a document called SPARS. I don't wanna say it might be like it might even be SPARS 2025 if I if I try to remember correctly. And, in that, they're talking about, release of a novel agent, etcetera.

And part of the tabletop exercise is them introducing a novel, vaccine platform. And the novel vaccine platform developing, you know, sequelae, particularly neurological sequelae, and, how they would plan to do that. And all of this is is in the open open literature. Right? If you wanna go looking for it, it's, and like I say, they've they've they've actually ran a very successful campaign because what they've done is they've managed to deflect and push back and kick the can down the road so that five you know, we're here five years later.

So the news of these athletes, etcetera, and the admission that, yeah, we are dealing with something synthetic is lost in the noise of current events as they, are unfurling today. But that the whole mess that emerged from that literally left tens of millions dead and probably orders of magnitude, injured and incapacitated. And, the the problem has been in trying to understand what causes, you know, the dead are the dead, let the dead bury the dead. But, for those that are, left with long term injuries, how do we, we need to understand what those mechanisms are and, maybe try to find ways to counteract what has been done. So, yeah, that's that's my introductory statement.

Yeah. And look and I thought, and it's lost lost us time. So and even now, we're still battling a lot of the various information that's coming out here. So what I thought I'd do, I'd just play couple of minutes of the last interview, and I'll get your thoughts on where you're you're

sitting with that right now. Mhmm.

They very obviously have understood that, okay, you you need the first primer agent to go around the population. You then have the secondary come in, which then bypasses immune barriers where nature has learned to evolve to deal with, these prion like, events. And this is a point I want to get to in a in a minute. But, they I would argue that they understood fundamentally how to line up everything, and that in totality is the weapon system. Yeah.

You can't just look at the virus. You can't just look at the, the secondary delivery mechanism, which is this gene transfection, assault. And you have to ask yourself, why were they so insistent on using gene transfection as the, go to remedy in this instance? They didn't have to do that, but they they did. And what they've what they've essentially done is that just just from, crude numbers, we know that approximately 13,000,000,000 mRNA type vaccines were delivered.

That doesn't include the other binds. And the, 13,000,000,000 approximately into 4,000,000,000 people. And so, essentially, you've got your weapon to cover half of the human population in that four years. And if someone was if that was a weapons attack, they can't now all they have to do is sit back. Right?

The job essentially has been done with respect to exposure. And, well, then you're gonna say, well, I didn't take the, the vaccine. It doesn't, doesn't matter in this instance because we've got a real world example of what spread of preons looks like by looking at the American white that white tail deer population. And we can see over the last twenty years that there is an exponential rise across the Continental United States and now appearing in other, countries. I wanna say reindeer population in above.

Any thoughts on that, Kevin? I think we went we've moved from theoretical to, more solid solid evidence now. Sorry. I switched my mic into mute. Yeah.

So, what was what was the theoretical basis for what I was talking about, last year? Which is that, you know, from my own analysis to published works, there was a recognition that there was an overabundance of what we would call amyloidogenic reactive epitopes on the, on the particularly the spike protein of SARS CoV-two and also the, what would be the manufactured spike protein from using gene transfection technologies as a public health measure. Now what we were or what I was thinking back then was basically based off I mean, they're they're one of the leading research groups in the world with respect to prions, particularly scrapy prions. And they showed, in a manuscript I'd have to pull it up real quick, but, this is the lab of Hammersch Sturm, Per Hammersch Sturm. And they showed that there is a 20 amino acid sequence in the s one segment of the spike protein, which, has a high catalytic activity for conversion of the normal prion protein into the scrapy form.

Now, I would say at this point a year on, that remains somewhat, hypothetical. There's, the well, the how would that manifest at sort of, epidemiological level? Well, there's a issue around cross seeding of what we call amyloid like peptides and Alzheimer's, Parkinson's, all these, neurodegenerative disorders. It's not necessarily gonna manifest as, Kreuzfeldt Jakob. There are reports of increased incidence of Kreuzfeldt Jakob, but, it's not.

I wouldn't I wouldn't say it's a tsunami of Kreuzfeldt. But in in the preceding time between when we when we last spoke and now, there's I've managed to acquire the unusual clots that have been, banded around on the Internet. And, unfortunately, for, these clots were received by myself from Richard Hirschman. Unfortunately, for Richard, where he was trying to raise an observation that he was seeing. Right?

I I honestly believe that Richard was trying to work in good faith. He was desperate to sort of, get this observation out. I'm gonna give him, I'm gonna presume he's he's every time I've spoke to him, his his his discussion with me is consistent and, he, like I say, gave samples out in good faith to people he thought were better qualified or who may have a larger voice in terms of reaching more people. And basically, these have turned into, you know, they're social media predators and are, geared towards, trying to get clicks. And the way they do that is to, push for the more lurid, ex explanation for what it is that they're looking at.

And this, again, comes back down to graphene, nanobots, hydras, five g, the usual, what I would call these are you know, I would make I would encourage people to understand that once biowarfare was deployed, whether it was deliberate or not, there was you're gonna get a whole series of, Distractions. I don't want to call it distractions. Strategic strategic planning in order to, obfuscate exactly what's going on. That's the whole point of these weapons. Right?

And I I would look at these agents and look at them I I tend to look at them as incapacitation agents rather than, instakill diseases. And, and so the I wanna say it's it's it's almost coming up to three years since these samples were first sort of being spoken about by, by morticians who were finding them in, in bodies that they were having to prepare for, burial. And it's it's a shame because, as a consequence, there's been a whole, series of absurdities built up around what the what they actually are versus what you can see with, the current cutting edge technologies required to to look at these, well, it's diseased tissue and it's proteinaceous and primarily misfolded fibrin. Okay? And, so I've received them and the, you know, the the people who were holding onto them or or trying to do analysis on them, they didn't have the facilities to just, even do, histological sectioning, etcetera.

And, I, you know, I do have those facilities and access to a whole bunch more that, can allow a really thorough probing of what this material might actually be. Now, and so what did what did I find once, I received these samples? So first of all, I did receive these samples, and you

know what? I might just I'll just go and grab, one just so we can just talk about it so we can, we can show that it's real. Just I mean, you can basically see anything under the microscope that you wanna see.

Like, you could, you know, put something under the microscope and then say it's Yeah. And audio or it's You know, it's data technology. But if you don't actually have the equipment to actually, improve the case. Yeah. And, you're you're seeing the same it's the same people often with, using sort of dark field microscopy.

They don't know what they're looking at. They don't know what they're talking about. And the thing is it there is an avid audience for that type of, interpretation of what's going on. Whereas, you know, the the actual base reality is bad enough. Right?

And so, if you're if you're in a situation where you're let's say, you the if there's a group responsible for this. Right? First of all, you wanna try and gaslight the public with the orthodox narrative that they tried to put out, right, which, you know, basically has sort of broken down and five years on, as we said at the beginning of this interview, it's, yeah, there were ill athletes. It's it is coming from a lab, etcetera. All the people that tried to call that out at the beginning and were shut down and, basically Absor and censored.

Yeah. Yeah. And I I got every example of that from accounts being shut down to essentially being debanked. Okay? This the tilt towards, I don't know, the nanobot hypothesis is is essentially their their last fig leaf from people or to to protect them from people like myself who can get hold of these samples and then analyze them properly.

And it's and let me just go and grab one, and then we can Yeah. I'll we can Okay. So, I don't know how close, but there Yep. There is one right there. I have 13, different samples from different, individuals who have been unfortunately, had to have been, dressed for, burial.

And the age range is wide from thirties to old age. And, what I what I can say is is that, one, prior to me receiving them, I had no idea, how much how much validity there was in what people were saying online. And, you know, I'm I'm gonna dismiss the more lurid stuff, but is there is there something that constitutes a a phenomenon to people people, although they're talking about it to some extent, that if it gets handed to someone like myself, I can look at it and say, okay. Here's what the properties it has. These are this is what happens when we begin the analytic process.

And Yes. This is a sample with a little bit of alcohol in there and then under argon gas. And, what I've been able to do now is say, I I'm confident now that, yes, they're real. I've had plenty of experience now in just handling them, cutting them, using them for different experimental designs, etcetera, and trying to find out exactly what the properties are. Now, before we sort of get lost in details, what I can tell you is is that this proteinaceous form is, like I say, highly it's misfolded, amyloidogenic fibrin.

K? And this is occurring in the blood of people. Now this is, you can consider this a separate disease pathway than the one I was when we were talking about last year, which is the potential concern about amyloidogenic attack, epitopes on the spike protein. And in this instance, we were talking about the hamstrung, preprint, which showed that it had a high specificity for, the prion protein and its conversion to the scrapy form. This is different to that.

But there is a, there is a commonality in the disease mechanism that between scrapy prion, misfolded peptides in Parkinson's and Alzheimer's, and what we're looking at here. And, what we have to try and find out now is what is the causal mechanism for the formation of these, misfolded fibrin in people's Let me I'm I'm I'm hearing what you're saying is that, correct me if I'm wrong, that last year we're talking about the, say, the spike protein causing a disease state. Now we're what you're discussing is the spike protein infects the body, and then the body is producing more spike proteins or whatever this thing is that is causing these clots. Mhmm. And therefore, I think I heard somewhere you saying that eliminating the spike protein itself from the body once the the process has been seeded, is not is is of no effect or may Yeah, mate.

That helpful. Might not be enough. So Yep. Just let's think about what we're looking at here, which is I took I've took the gloves off, so I don't wanna handle them, too much. But I'm essentially receiving, samples in the tens of grams in weight.

Okay? And people's exposure to spike protein is gonna be in the picogram level. So this phenomenon is, I would argue, a self catalyzing reinforcing phenomenon once the, the initial seeding event has happened. And this is again, this overlaps with what we understand with amyloidogenic prion like disorders. There's a seeding and then the there's a amyloidogenic cascade, which leads to the manifestation of the disease as we understand it.

And so So at this point, what I've been doing well, actually, let me sort of take a step back. So prior to receiving these samples, which have come from cadavers, What I was trying to do was to take the, the spike protein itself, and it's possible to buy spike protein. It's called technically it's a recombinant peptide, which is, it's kind of similar to how the gene transfection worked with, with respect to vaccination campaigns. But what they do is is they'll have an expression system, usually bacterial or, there's a common, cell line called hex cells. And you can you can force those expression systems to make you a whole bunch of spike protein.

And what I've been doing is, well, for a year or so, I was setting up the lab and, at the same time, trying to work with a technique called RTQUAIC, which stands for real time quaking induced conversion, which basically just means you have a well plate 96 well where you put samples in and then you, you shake it, violently for thirty seconds and you look to see if there's any buildup in fluorescence where the dyes that we use to detect, amyloid aggregation, would bind to any products that are for forming in your tray. And that that gives you some idea as to the amyloidogenic prion catalyzing potential within those peptides. Now

I I was able to well, let's just say the, using the recombinant peptide approach, the results were ambiguous. Although I got some positive hits, the hits were weak and, not a so, where was I? Yeah.

So the in terms of recombinant peptides, trying to, well, to recapitulate what, Hammerstrom was talking about, I was having very little success and very little success with an animal model platform in which to test. So I was having very little success, in that regard. And it was frustrating because you're, you're well, one, we're not, we're not a funded government institute. Right? We're we're Yeah.

We're trying to do these experiments of a, very limited budget and essentially my, time and commitment and, well, ever expanding, strings to my bow as I, try to develop techniques to examine what it is that we're, looking at and seeing if we can, re Dropped my sample on the floor. Hang on one second. From a hypothesis that the spike protein produced these, the spike protein potential. Right? Yeah.

So that amyloidic amyloidogenic potential is there with the virus and most likely there with, people who receive the vaccine. In that, in that, space, we have to because the institutes have been so inherently dishonest, we have to find what are reproducible phenomenon and, reproducible enough to allow testing of countermeasures that what we're trying to do is basically open source and your cure. Mm-mm. I wouldn't cure is a big word. Treatment is a yeah.

Yeah. Treat treatment because, you're you're dealing with a complex, disease process. There are no cures for, amyloid disease states. There are now if people sort of cast their mind back at at the beginning of SARS, there there was a, the FDA rushed through, some antibody based treatments for amyloid type conditions. And there was, in fact, some some pushback from, well, officials who were on those review panels and some resignations because the work wasn't there to demonstrate efficacy, etcetera.

There's, you know, there's a lot of work and, unfortunately, a lot of work going into trying to treat these disorders. But just even if you think about something like Alzheimer's or Parkinson's, if that's an amyloid type disorder, then it sort of falls under this umbrella and pea and people are trying to find ways to mitigate and control it just to, give a give a few more years quality of life, at the end of, people's time here on this earth. And, unfortunately, a lot of that is just about milking the, the wealth out of people at the end of their lives as well because, you you know, if you've got the cutting edge treatment that's sort of rolled out of these laboratories that has the, the patent on it and they can basically charge what they want. And as people sort of go through the, the unfortunate terminal we all have to face it, but the terminal, trajectory, the system, particularly in The US, is set up to sort of extract wealth out of the poor unfortunate people who end up, going through these, treatment modalities. Now do they help?

Potentially. Would you want to have them if you're suffering? And, does the money mean so much at the end when you are suffering? I can see why the system is set up in the way it is. But I would, I don't like it.

I just have a I have a visceral dislike to, that type of approach to people's care. And so we've been looking towards trying to find, yeah, just open source, non patent interventions that people could take and, you know, if if they work, great. If they don't, then, back to the drawing board. But the and so to sort of get back to the original question, which was, you know, last year where we were talking about hypotheticals, at this point, we're now being able to look at concrete not concrete in the literal sense, but, actual tissue that has been described by individuals as being unusual in its appearance in terms of timing. And the the claims are that, the, the people who are first seeing these I don't wanna say first seeing them, but are seeing them where they say they weren't seeing them a few years before, now trying to figure out what to do with these samples as they try to as it makes their job more difficult because they can't, dress the body as they normally would because the vascular system is blocked with these types of proteinaceous, obstructions.

And so, yeah, it's, I wanna say fortune has shined on us because, it's just so unfortunate, the source of where they've come from. But now we basically have a huge evidence almost. Yeah. Well well, yes. It's that's something we can get to, later on in the discussion because this is, it it's important factor that we need to think about.

But the, in terms of having a source of diseased tissue, right, where I've spent a huge amount, not only of time, but, in in financially trying to like, these recombinant peptides are not cheap. Right? It's a thousand dollars per hundred micrograms. Right? And that lasts, that lasts a couple of experimental sessions.

And so, where we were or I was struggling to sort of, get something reproducible, now we're in a position to be able to say, okay. We have this tissue. What do we see from the macro gross anatomy to the, micro anatomy to the molecular? And, unfortunately, for in terms of public health, this, all the evidence points to this being amyloidogenic in nature, and I can walk you through the slides and the an the analysis that we've done that shows what it is that we're dealing with. And I don't know if you wanna Have you actually managed to test any, of these clots or whatever your cloud columnaric clots that have come from, the living?

I can't have it in a blood test. Has anyone managed to send you that? Because I know that, you know, you you see it anecdotally various people posting that they've come up. But, obviously, we don't know what they are because they haven't been been tested yet, if they're exactly the same as what you're seeing. So so let's let's sort of set the record straight with, white fibrous clots.

White fibrous clots are part of the landscape of phenomena that is, occurs in the cath lab and surgery when people turn up at a hospital and are suffering from stroke, blocked arteries, blocked let rate. And so the apparently runs to about fifteen percent. Now, what I you know, there's there's been a lot of discussion about, well, why aren't people seeing these or reporting these in the living? And I think the answer is is that post COVID and the vaccines, just the system has absorbed people turning up with these clots and are basically just they're probably just putting it down to this is the new world in which we live in, which is we know that COVID and they admit to vaccines having the primary, adverse event profile is cardiovascular and, thrombotic events. Okay?

So I think what's happening is is those cath labs, etcetera, are seeing these phenomenon. It's just put down as, yeah, it's the world we live in now. It's COVID related. They certainly aren't gonna be looking at vaccines because it's just not worth, the trouble for them. And it they'll do the treatment.

Probably the clock goes into clinical waste. And unless there's money and an active research program to be looking at these clots, then there's, there's no reason for them to start dissecting them and trying to understand the actual nature of them. Right? Yeah. And that's unfortunate, and and and it just rides on the fact that white fibrous clots have always been with us.

Okay? And, But you're saying this is this this thing is quite novel, what you're seeing within these deceased bodies. No. Let let me be very clear here. Right?

I'm neuro I'm a neuroscientist. I worked around misfolded peptides that occurred within the central nervous system. I've I have zero experience in cath labs. The closest I get to working with cardiovascular systems is, termination experiments where I have to extract tissue, and that involves perfusion of where I work with primates. You have essentially it's trans cardiac perfusion is the technical name for the procedure.

You open the chest like you would, in an open heart surgery, cannula inserted into the, into the heart, and then I'll pump out the, the blood and pump in formalin to preserve the tissue ready for, histological processing. And in that process, I see a whole bunch. The blood will clot as it comes out of the body. They're typically these red jelly like, clots. I don't I don't ever remember seeing, white fibrous ones.

I and white fibrous clots are or generally, clots when people go to the cath lab, I think, generally, there's something's been building up over years, If we think prior to COVID, that is and eventually some sort of catastrophic state is reached, they go for a treatment, emergency treatment, and, you know, such that's the framework in which we were always working. And, I I think that because any anyone who's sort of responsible for the, the surgical procedures is just going to is going to look at what comes out after they've done the thrombectomy, which is basically mechanical, extraction of these clots and just put it down to

yeah. It's it's that form versus this form. And, the again, it comes down to, is there a active research program looking into these clots to say these are different to clots that were retrieved previously to COVID. Now Yeah.

I would I would very much like to get my hands on a pre 2020 series of white clots to see how much they, overlap with what I'm looking at here. I think what's unusual about these ones is just the, the size and scope of them and the fact that, if the morticians are booked, we'll just give them the that they are telling the truth in this instance. And but it's it's also a possibility that, just because of the, well, the unusual societal dynamics that took place as they shut people down and forced mandates, etcetera, that maybe, you know, what was just ignored previously suddenly gets attention now from, morticians. That's that's something that Yeah. We have to sort of, hold in our minds as well.

And so all we can do at this point is forensically examine this tissue, like I said, from the macro Mhmm. Down to the micro, anatomy, to the molecular, and then see if there's anything that we can, tie back to, the causal agent. And I like I say, just to forewarn people, when you're dealing with something that potentially where you you had a catalyst in the picogram range and suddenly you're dealing with, masses of tissue that comes in at tens of grams, it's probably gonna be difficult to find the causal switch because of this self catalyzing property. Right? So there's a seed formed in terms of abnormal clotting, and no one is gonna argue the case that in when you get exposed to COVID or you get vaxxed injured, there's a demonstrable phenomenon of amyloidogenic microclotting.

K? So people will, unfortunately, they just there isn't an automated way to do this type of, investigation. It literally requires someone manually looking through a sample and dyeing the sample for amyloids with the, the accepted, validated dyes that are used for this type of study. And basically, just counting, how many objects that they see, in someone's blood sample. And if so, you know, just to give there's an article called it scuttlebug, but they're like the discussions that sort of go on, in with people who I know are working in this space is that now it's more and more difficult to find people who just have, normal blood.

Literally literally everyone and but the problem is is that when peep the the samples they're looking at is people going and trying to get treatment because they don't feel well. Copic shoes. Yeah. Yeah. And so we do so it's difficult to say We don't know what the population wide distribution is of Right.

Like, we are we are assuming. Yeah. And this is what you've done. You've you basically got the, the samples, from the cadavers, and you've sliced them up and put them into, a machine. And I think probably the most concerning part was that in seven hours, you were able to get these it to to show that it was amylogenetic, that that it could be potentially seeding.

Like, let's say, someone was exposed to to that somehow. Yeah. That that could develop some sort of preogenic disease. Is is that what I'm I'm seeing? Let's let's let's work through the data.

So I will try and do a, PowerPoint presentation. Right? And, we'll work from there. I just wanna make sure I'm pulling up the right one. And just a quick question.

Someone asked you with Alzheimer's disease. Is Alzheimer's and considered a prion disease, or is it Yeah. It's an amyloidogenic disease. And in fact, it's it's one of the canonical ones because, in addition to, misphosphorylation of tau, you you amyloid is the it the amyloid beta and, its conversion through gamma secretase is the one of the primary, mechanisms that's been identified as being correlated with the disease. Now if you wanna get into the weeds, there's, been some debate as to the veracity of the original claims around amyloid and the, western blots that were produced.

But I think the overwhelming weight of evidences is that, amyloid misfolding is a big issue, and, you know, I'm I'm not gonna that's a that's another research. Yeah. And then, of course, there's, Karoo disease that was found in Papua New Guinea, which somehow died itself out. That's that's a canonical prion disease, right, which is, very, very similar to, Creutzfeldt Jakob. It did have a slightly different presentation.

Again, there's some, some debate as to the causal nature of it. Some people claim that it could be due to vaccination campaigns that occurred during the second World War. Again, those those are a whole it's not that they're invalid areas of, investigation. It's quite fascinating. I'm I I love speaking to people who have taken the time to, to go through, the history, the anthropology of those events.

And it's important that we, we understand the history of how of how the disease emerged, how it was named, and the people involved, etcetera. And, you know, the individual who first identified Kuru was, basically went and plonked himself down in Fort Detrick and worked in the bioweapons programs of The US. Right? It's the you know, there's And and and I and I hate. So, you know, but they could have possibly used Karoo as a, testing ground, which they love using islands for because it was nice and in a nice sample there.

Yeah. And to develop and buy weapons from Yeah. And look. The simple fact is prions have been an active line of research in biowalfare programs, and, that should concern everybody. Right?

And and and it's the problem is you have to you really have to develop a nuance of thinking, which is that it may not be scrapey that they're aiming for. Right? If the and maybe what they well, what I think they're aiming for from a biowarfare perspective is incapacitation. Right? So these are meant for battlefield effects.

You wanna you wanna knock out as many of the opposing forces that you're going up against, and you wanna find a, efficient way to do that. And an efficient way to do that is one through, highly transmissible pathogens that contain these, reactive epitopes, and you just try and load up as many reactive epitopes as possible into a synthetic peptide. There's other ways of just spraying, contact, toxins, etcetera. There's there's innumerable pathways that they look for to try to see how to seed the environment with these peptides in order to obtain an advantage on the battlefield. And the Yeah.

Terrible fact is is that incapacitating agents, meaning that if they don't kill, are perfectly fine and legal under all the, treaties and, agreements between, the big power players. And, that that I'm afraid is the, unfortunate world in which we, find ourselves. It's and, you know, I I can't speak to how much of this was deliberate versus how much was they just something went wrong. They had a they did have an accident, and then we, we end up where we are today, which is, a very, very large disease burden. And, the authorities being less than helpful in dealing with the situation because, one, the interface with the public is gonna be this is unfortunate, but, a lot of doctors are in debt because of medical school.

They're often very ideological in their, their thinking, in terms of the the power dynamics between them and the patient. And, there's there's all sorts of obstacles in the way to getting the, the clearest answer, which which I think the public is owed at this point. Right? And after after they've lied about the origin, after they've, essentially engaged in mass, psychological operations where they, quote unquote, lockstep people into a public health measure based around gene transfection. And suddenly we've got a, emergence of, you know, disease forms that because of the nature of the disease causing agent, again, it's just gonna manifest in people differently.

And you're gonna have autoimmune disorders, cancers, cardiovascular, etcetera. All of those can have a proteinopathy, meaning a misfolded protein as a as the causal part of the disease that's manifesting. That didn't sound too convoluted. And then people will not know the point of origin of it. Right.

Right. We're working on now that if we find the point of origin and the mechanism for how these things are being manufactured in the body, we potentially are looking towards some relief. So hopefully. Hopefully. Now, look, I can I can say that, with some confidence?

Now I'm I don't want to I have to maintain scientific, neutrality in in the current environment. Okay? But I'm fairly confident in the results that I am seeing in the groups that I'm collaborating with that there are techniques available to lower the burden of, spike protein, which does seem to be a factor in, long COVID vax injured people that they see there seems to be a reservoir of spike protein. It's and the body is reacting to that, and it's not just it's not just, immune dysregulation at this point. The peptide itself, as we'll see, is causing, this buildup of toxic peptides in the body itself.

And though that that buildup of toxic peptides potentially leads us to a good friend, here, this amyloidogenic clot. Okay? So I'm going to share screen. Is that available? Let's talk about, what my lab has received, and, I wanna I wanna be very clear.

During COVID, I have held a number of positions. One, its origin is synthetic. I've worked very, very hard to dismiss what I consider psychological operations with respect to, there being graphene oxide and nanobots inside vaccine vials. And the same techniques that I use to examine the vials then, Raman spectroscopy, scanning electron microscopy, form part of the backbone for looking at these, peptides here. Okay?

And I wish to distance myself as much from the orthodox institutes that gaslit the, the globe as a whole versus the grifting side of the social media networks that are looking for well, they're they're just making up lurid BS in order to attract an audience and, basically basically maintain a, funding. Well, that's not the case. The wrong wrong, yeah. But often they're pushing, vitamins or they're Yep. You know, it's it's the it's really the it's a over exaggeration of what is the base biology.

And the best example I could give of this is the, with the spike protein as it first emerged with COVID, there was an observation around there being things like homology between conotoxins, which are, toxic peptides from sea snails, and snake venom like peptides, which attack, cholinergic receptors. They basically irreversibly bind these receptors. And once that happens, your, your autonomic nervous system basically gets shunted into, mass dysregulation, and I can tell you that was one of the standout features which I had. But that that phenomenon in and of itself doesn't support people going around saying they're putting snake venom into the water, Okay? Which which is one of the one of the issues that we have had.

And it's it's that side of the Internet that I I don't want anything to do with them. Right? If you put me on an interview with them, and I'm just gonna sit there and and just destroy them verbally and scientifically, because I have to. Right? Yeah.

Because I want to make sure that people get the most That's true. I want to it's not truth. It's it's it's as objective and rigorous scientific analysis as possible. Yeah. Okay?

That's all that's all I'm aiming to do. Okay? So, what are we looking at here? So, on the left hand side, that's the arrival of the first samples that came to the lab. Same patient.

One on the left is just preserved in a little bit of alcohol and argon. The other, is coated in formalin, and we're gonna start by just looking at the gross anatomy. What happens when I crack open the vial, pull it out, and what what does its physical properties feel like to me? Okay? And so, I've done this live, on on stream.

Right? People can go back and look at the streams that I have done on my channel, and you can watch in real time as I do this and, do the, do the analysis. Now I I I can't do everything all the time under camera, but I try to do as much as possible. And, just as a heads up for people, just because of the amount of time that I'm having to put into the lab, it's it's too much for me to set up a, full stream, like, kind of rumble, etcetera, to for people to watch what I'm doing in the laboratory. But I do what I do do is using my Discord community, I will go live in there, and then everyone who's joined in there are more than welcome to watch me working in the lab.

Now, people seem to like it for some reason, and I'm happy I'm happy to show what I'm doing, which is just, you know, opening up indoors, you know, taking stuff off with, pipettes and, you know, just running stuff. You know, there's some work on the animals that I've done, and but I can't do I can't make a full stream out of that. So if you wanna see those details, ask to join the Discord. Okay? And, you know, that's first first point.

So so, yeah, you're looking at a standard, dissecting microscope there and, basically, it's a pair of forceps and, essentially, hypodermic needles that I will use, basically, to pin the sample under the objective lens so that we can look at the, the object at hand. This is not through the microscope. This is me just taking a picture with my cell phone, but now we'll start to look. Well, let's talk about what it is that I see when taking that sample out. And, yes, it feels rubbery.

And, the closest that I would describe it in terms of how it feels is I would if people have ever eaten, like, crab sticks, fake crab sticks, it feels like that. Right? But maybe a little tougher because a crab stick sort of peels easier. Okay. But in terms of tensile strength, it's close to that.

Now I it the although there's resistance, I could pull that clot apart if I wanted to with my fingers. Okay? It's it's but it does have a degree of elasticity to it, and, it does appear unusual just in its presentation. Okay? Just just to my eyes and just, my, experience working in the life sciences.

Okay? So, this should say one millimeter. I'm sorry. And this is, now looking through the scope for dissection, and you're just getting a close-up view of the surface of the clot. And, you know, the, what would I say looking at that myself?

There seems to be a striated appearance to it. Right? So I don't know how clear that is to people. It looks pretty clear. Right.

And there are also sort of major creases and folds. So how how would you have expected something like that to look instead? Look. The the only clots I've ever had to deal with before are just the immediate ones after cardiac perfusion. It's usually the jelly like clots that literally would just, you know, if you've handled them, just sort of break apart very, very quickly.

Like I say, this this is proteinaceous and, you know, it's it's almost, the rubberiness of it is almost like the brain after I've done a trans cardiac perfusion with saline and ran some formalin through it. Mhmm. That's that's kind of how it feels. Right? But, obviously, the brain being more, lipid in nature, it it has a, more spongy feel.

But, in terms of sort of tensile strength and just sort of feeling it, fingers and what have you, think crab meat or if you've ever worked with, perfused perfused tissue and, brain tissue. There's it's close to that. And, you know, obviously, the color is important, because the color will tell you some idea of, its constituents. And the normal or or the majority of clots that come out are are ones that essentially are aggregating red blood cells and platelets and are loose well, if they're immediate rather than sort of impacted bonds over time, the fibrin is only, just having chance to form, and so it's not forming a more, solid, mass than, that I would usually see. So I see in my experience, I've seen immediately forming clots.

I I don't have experience in the cath lab pulling out a clot that's been building up over decades. Okay? So While we're talking about the clot, are you in agreement with the analysis that it has high levels of what was that? Phosphorus? Phosphorus sulfur.

So I'm lining up these the I know other people who have done the ICP mass spec. K? I'm not gonna say that they were wrong in their analysis, but, what I wanna do is just I'm gonna do this as independently as possible. I will run all those tests and more to try to, determine exactly what it is that we're dealing with. Okay.

And, what I will say is that where I've done mass spec type measurements, I have not found anything that would point to there being metals, tin, heavy metals being, part of the, picture. Right? It's it everything just looks not just. Everything looks like protein. And in this instance, in order to work out whether you're dealing with a misfolded protein, there's a whole number of steps that you have to go to, through in order to say that you've ticked all the boxes to say that you're confident that it has the properties that we ascribe to misfolded peptides that we've been looking at for decades, okay, and associate with different diseases.

Now, so to your question is, is there a, overabundance of phosphorus and phosphorus plays an important part in the body, your, so there's a metabolic pathways called kinase signaling, and control of phosphorus along those pathways is critical for maintaining bodily health. And to give a sort of example that relates to what to COVID, one of the earlier more spectacular observations was that, cells infected with COVID began to take on very different cellular morphology than you would normally associate with. Let let's just say we're talking about lung tissue. Okay? Yep.

So, cells within the lungs get infected by virus. Viral infected cells undergo morphological changes in which they develop pseudopodia, which basically means that they develop branches and tentacles. And that switch in morphology is, a process or dysregulation of

kinase pathways, and that involves phosphorus signaling through multiple, multiple steps. They're incredibly complex pathways that essentially, in this instance, are interacting with actin and which forms the at the at the cellular cytoplasmic structure, if you like. Right?

So a cell a cell has actin in it, and actin helps maintain this the shape of the cell. What's happening is the kinase pathways are changing. Actin is forced to change, and it then starts making these pseudopodia. And the pseudopodia act as an attack vector for viral vesicles to, travel to neighboring cells. Right?

There's there's fantastic work out there, Harrison, and it's going back a few years, but where they literally they literally can show viral particles traveling and long and through these pseudopodia tentacles and piercing and infecting neighboring cells. K? So, does does that relate to what we're seeing here? I'm I'm unsure. Again, causality in interrelations is a complex, complex topic, man.

You you can spend decades trying to work out something like a problem like that. Okay? So I don't I don't have answers in for this particular issues. But, let let's let's just give them the credence and say that the ICP mass spec that has been done has found a, large phosphorous signal. Beyond that, I'm not sure what I can say or what or what that means really with respect to the final form here, which is the amyloidogenic stacking and misfolding of protein in this instance.

Okay? Because I I haven't done the analysis yet myself, and I wanna make sure I've run all through that through all samples, and then I'm gonna have a better idea of, what we're what we're dealing with. Okay? And that that's not to that's not to dismiss the work that others have done. It's just I You might as well confirm it.

Yep. Yeah. I I wanna be clear in my mind, and I wanna I want to have done the experiments just so I know what exactly what's coming out. And, and I I'll have it done in the coming weeks to months. Okay?

But we have we already have enough to sort of, move on from there. So, we're looking, yeah, the sort of gross morphology here. The, you know, obviously, the color is different to normal clots. It has this, striated appearance. And I think a lot of these sort of larger, morphological features, I think, just come from the, imprint from the, inner lumen of the vessel in which the the clot is forming.

Okay? So what we're so now just I'm basically just taking and if you go back and watch the videos I don't know. What's this is up here? I need to change this. And I'm taking a scalpel blade, and I'm just slicing through that, segment.

And what you can see is that it's not a, tubular structure. So people have claimed that what the morticians are pulling out are blood vessels, etcetera, and this shows that's not the case. These are, it's a solid deposition of peptide that, well, even saying peptide prior prior to me

looking at it was, peep people would have challenged that because, the the test hadn't really been done and the people doing the testing, but they would say probably weren't qualified to be doing it, etcetera, etcetera. Okay? I'm not saying that, but it's just the, the criticism that's been levied, at people who have tried to look at this.

And whereas whereas I think Richard has been, look, Richard is quite, humble and honest about when he talks about this issue, which is he says, I don't understand the techniques being deployed. I, again, I handed samples off in good faith, and the problem is is that people have taken those and, just ruined, the scientific Yeah. Investigation that should have taken place. Now had he been able to reach me two years ago instead of, those, you know, the Jane Ruby, GRIF network, and what have you. We'd we'd be much further down the road to understanding what these are without the without the, Noise.

Yeah. Noise is, is, I don't think it's quite the euphemism that we need. Yeah. I kinda be diplomatic here, but, yeah, I mean, I think the the problem is is we're fighting these search engines and the the the shadow banning and all of this. It's it's very difficult to, get this out because all the people that, have the scientific knowledge, it it seems to be downgraded like yourself.

You know, you you're dealing with all these bans, which is why it's important that people take this video, cut it, share it, do whatever you want, try and link back to, you know, link back to your website. So because we actually need people to do that because we can't rely on the algorithms at all. No. And you've got you've got active organizations, right, that were that were literally funded by the World Health Organization, the UN, USAID, you name it. They were pumping millions and millions and millions in to control what people were saying and whether that was banning people on, I thought, on Twitter, to, literal media campaigns across TV, magazines, etcetera, and, talk shows, you name it.

Right? There was a full court press in order to shut down people's, questions about what happened. And the simple fact is, we know for certain now it's synthetic in origin. The, the campaigns to push for the public health response were highly coordinated. The fact that they were so similar across the globe tells you that it was, an org organized from a central node.

Where that was could be any number of places, but, you know, think military in this particular instance. And they're gonna they're gonna obfuscate themselves through quangos and all manner of, well, the the the lit that they'll put grant money out, and you'll have these organizations go for it in a feeding frenzy. They'll set themselves up as some sort of, you know, independent group of scientists and but they're receiving money from government, from, I'd say, you know, USA being the obvious example in recent months, and they're gonna tow the line with respect to telling the public what it is that they should be thinking about this phenomenon. And, it was all lies, right, right from the get go. And those those of us who were qualified to speak up trying to say something was squashed aggressively.

Right? Yeah. And, you know, how you responded to that was, down to yourself. You know, I have my own way of doing that. So I don't know.

It seems to have got me this far. Yes. The and and now the sort of scientific work has begun where, you know, I spent I spent way too much time looking at vaccine vials to convince myself and people listening who had questions about those, products that, they're yes. There's some industrial contamination that's you can potentially find in there. It's primarily metal in nature.

There's no, again, there's no nanobots. And what people think are nanobots are essentially just crystalline forms of cholesterol, primarily. Yeah. Right? So and cholesterol makes up the, primary bulk of the lipid nanoparticle.

Right? PEG cholesterol, etcetera, etcetera. Anyway, moving on. So it's just another look. Let me just go through this and change this.

And, and so that's that's the gross morphology of just one sample that I've pulled out. So, it it already looks different from a what most people would imagine a blood clot to be, which are usually sort of red in nature. And, you know, again, depending on the age of them, their density and, stiffness, will vary. Again, it's very the the color is off here. I think it's just a consequence of my camera and the, angle How how wide are they?

But how no? No. It's because you've got the scale bar there and, yeah, sort of two, three, four millimeters wide, two millimeters thick. And I think it depends the vessel on which they get. Again, I'm getting them in, you know, sort of 10 mil tubes.

I've seen much bigger, shown to me on camera. And but, you know, this is enough for me to work with in terms of doing this type of anatomical analysis, molecular analysis, and testing against, or trying to use it to develop a test platform in the animals. Okay? Well, certainly, they have no place in the blood. Right?

Oh, yeah. For sure. Someone someone who like, the concern at the moment is, is there a, silent wave of people carrying around this, this blockage or the the this this clock like phenomenon? And, again, I would go back to what I was saying earlier is that what I think has happened is is the the system has absorbed it. It's basically, recognized and stated that, yeah, we know that there's a clotting phenomenon.

You know, the primary diagnostic criteria for COVID is systemic coagulopathy, meaning you've got clotting occurring across the body. Okay? Now that, in a lot of cases, looks like the microclots, the amyloid microclots than usually a hundred hundred micrometers in length. Okay? Sometimes bigger, sometimes smaller, but on on average, about a hundred micrometers.

These larger clots, again, I I think have been absorbed by the system. And if there's if the powers that be worked so hard to obfuscate to the public the source of what COVID was and the nature of the public health measure that was enforced upon them. You don't think they're gonna be just putting the brakes on in the surgical centers and cath labs that receive these patients and basically just saying, look, man. Just make a note of it and then just throw it in the trash. And if someone if someone is unfortunate enough to be severely injured or dies from that, that basically gets labeled as a cardiovascular event and potentially COVID linked.

Right? For sure, they're not gonna be saying anything about vaccines in this instance. Okay? Now I'm going to I'll state for the record here. I'm gonna I have to remain agnostic as to the cause of this phenomenon because it's clear within the literature that spike protein in and of itself can form a or or or can initiate and catalyze a misfolding of fibrin by itself.

Now, but I would say, does it help if suddenly, you you expose people and dump in billions of molecules of reactive epitope that we know we now are working towards it causing a misfolding of fibrin, a a pathological misfolding of fibrin and the clotting process. Right? So why are these forming? Because of the misfolded nature of the peptide, enzymes that would that would be involved in their breakdown, like frombin, are unable to latch onto them and begin the degradative process. So in effect, they'll hang around.

And because of the type of disease mechanism that we're dealing with, which is the amyloid templating of normal healthy peptide into the abnormal form such that it sticks together and then begins to aggregate into bigger and bigger structures. Now what's the basis for that? What it's, the current hypothetical framework is and I I think there's a lot of evidence for this. Some it's not new in the, the fields that I work in. It's that the, there's a development of, what's called, beater sheets that become hydrophobic.

And those hydrophobic beater sheets will then stack together, stick together like Lego Lego bricks, and just form larger and larger fibrils. Okay? Okay. So, this this was basically the extent to which people had looked at these clocks. Right?

That like I said, there'd been some mass spec analysis done, but the, rigor required to look at them beyond this hadn't been applied. Okay? So, in my lab, we have the capability of doing all the histology and more to look at the, the microstructure of the microscopic structure of these, of these clots. Okay? So, how do we do that?

This on the left is called a freezing microtome. And what you do is well, there's two types of microtome primarily. One is paraffin fixed and embedded, where it's a that's a good way of, quickly scanning through a section, but it's not so good for antibody work. I went for a freezing microtime because I thought I was gonna be doing far more antibody type, anatomy with respect to the work I was doing with recombinant peptides and any, modeled type pathology that we could induce in animals. Okay?

And, you know, the best laid plans of mice and men, as such. I've had to I've had to pivot around, the original aims and directions that we're going for and use the setup that we do have to go, to try to, analyze these, this phenomenon. But what this machine does is basically, it'll hold the so you see the orange block in the middle on the right here. Right? So this will go down to minus 30 degrees Celsius.

It's held in a special type of gel, and the there's a very, very sharp knife, and it will step forward a set number of micrometers every time I wanna cut through the tissue such that you get even slices of that tissue so that you can put it under a microscope. Okay? And that's what you're looking at here. So I've basically taken three slices and put it onto a standard microscope slide. Now the thing that I would what should stand out to people is what look like here, what looks like whole tissue.

Right? Oh. When you start cutting it at the level required for microscopic investigation, you see cavities and, small small holes that are visible to the naked eye. Right? So, that's essentially now what we're gonna see as we start the, process of going down, in scale from, you know, the light microscopy into, the realm of electron microscopy.

Okay? So this this is, at four times this is generally the lowest objective magnification that you get in a on on any microscope, whether it's a $30 kids microscope off, Amazon to, you know, the microscopes that cost thousands and thousands of dollars and can do all the fancy tricks with UV lightning. And so, what do we see? Yeah. There's, it's not a solid, plainer piece of tissue like you would see if I was cutting, say, brain or, muscle tissue or any other any other organ tissue, in the body.

It would be very, very unusual to see this type of, porousness, I would say. Yeah. I guess I guess that works. And, those with a keen eye will start to notice that, there's a, fibrillar nature to the to the material. Right?

And this this has been cut at 20 micrometers. Okay? That's how thick the section is. And so this this is now at 40 times magnification. That's actually the maximum that I have on this scope for UV investigation.

But it's quite clear when you're looking here that, yes, we do see fibrillar type structure, and, this this would concord with it being fibrin. Fibrin forms fib fib five fiber like networks that are basically designed to hold platelets and red blood cells into clots. That's its job. And you have to think of, in biology, sort of, form equals function. Right?

So its job is to form a, network that acts as a sort of trap for normal clots, and the that's you need that so that you don't bleed out should you, I don't know, step on a nail or, you know, whatever. You know, there are if you're a hemophiliac, etcetera, paper cut can be deadly, but, you know, you need a clotting response, and fibrin forms part of that. It's it's one of the major

components of your clotting that's occurred here. Now what's, of interest here is that there is very little or no to identifiable red blood cells or platelets or any of the other normal, tissue types that we would associate with a clot in this instance. Right?

It's just this very fibrilliform, and the, the color that you're seeing at this level, which is just normal white light, being shone through the, specimen matches what you're seeing at the macro level, which we looked at a couple of slides previously. Okay? Now okay. So this So is this is this stained or not? No.

No. So this is what you when you actually cut it and you put it under the microscope, it has that green appearance. It it's it's not green per se. Like, to my eyes, it's more a, it it's a cream color. The green that you're seeing is refraction of light through the fibrils because essentially they're translucent.

But if you look at if you look at more dense areas, it's essentially more akin to the color that you're seeing in the in the larger samples. Okay? So, so with light microscopy, you get some idea now as to what the structure is that we're dealing with. So we've gone from, gross morphology down to the, you could go to a hundred times with light microscopy and you'll get, you'll get a blurry view of, some of those some of those fibrils. Okay?

But what we wanna know is is what's the what's the nature of the peptide chemistry that makes it up. Now a way to do that is through all manner of different stains, but because I've been banging on about amyloid peptides, prion peptides, etcetera, etcetera for years now, I've been asking, and it's it's how I managed to get a hold of these specimens was just by people who were working on them. I said, you need to be doing amyloid staining on those tissues because you can't say anything, about their functionality until you do that. Okay? And that's how that's how I sort of managed to get in contact with Richard and get the get him to send samples.

Okay? So, what do we see when we look under the specimens with so you can switch to ultraviolet light, and that's how we use well, the primary dyes that we use. So the the legacy dye was something called Congo red, where you would have to look at it under sort of polarized light. And if you see and the problem is is that that effect is subject to a subject no. It's prone to a degree of subjectivity and because you're looking for something called bifringence, which basically says, under polarized light, do you see a halo around the structure that you're looking at?

And if you do, that would indicate that you're looking at an amyloid like structure. Okay. Using more well, I say more modern, but they've been around for a long time. But the other approach is to use, analysis under UV light and look for, absorbent spectra and the response to specific dyes that, have a an affinity to, amyloid structures. And so what does that mean?

So I talked about the beta pleated core that we think makes up this, the primary structure of the amyloidogenic peptide. And what these dyes do is between each layer of the beta pleated sheet, they'll they'll sort of insert themselves, and they'll go in there, and they'll fit fill as many spaces as possible between each layer. And then under the, UV light stimulation, they basically change, confirmation and will basically give off a, a a wavelength that's, wait. Hang on. I have to do that.

So if you if you if you hit it with four eighty nanometers, I don't I'll say they they they will reflect light back to you at four fifty, I think. Something like something like that. I have to have to go back and just, refresh myself on the details there. But, and so, one of the first things that I noticed was, if I just looked at that tissue under, UV light, so that's what you're looking at on the right hand side of the screen, it auto fluoresced even without putting any dye on. Now there's a there are some molecules in the body when you're looking at tissue that you'll see when using UV microscopy that you'll that will light up, and we know that they exist.

Okay? And, I don't know, is a very common one that you that you will come across. And this but I've never seen whole tissue sections without staining light up to UV light like you're looking at there on the right hand side of the screen. Right? So it's basically saying THT fire flavin negative.

Right? This is at the lowest magnification and, doesn't without doing anything to it except cut it into those 20 micrometer sections, putting it under a microscope, and then hitting it with UV light with a blue filter that will then excite in the green range. K? When you drop fire flavin onto the slide, onto the specimen, suddenly what you'll see is that specimen will avidly take up the stain that you start to see far more features begin to appear in the image that you see off the microscope. And what you can see is basically at this at this level of magnification, you're looking at small sort of punctate dots all around the, the sheet of material that's been taken from the microtome onto the slide.

Now we can sort of get into discussion about exactly what that means, but what I would my first and the way that I look at it is is that when you when the amyloid is forming, right, it's gonna start stacking. And where it stacks at areas of high concentration, you're gonna get higher concentrations of dye. And that's that's what I believe you're looking at here is all those little dots are areas where you've got more, stacking relative to the more lighter areas of the of the image. Okay? I know that's a lot to sort of throw at you.

Do you do you have a And the implications of all this? The the that you're dealing with a any systemic amyloid type disorder is considered along the sort of scale of, disorders that you you can turn up to a hospital with as being one of the more serious conditions that you can develop. Right? And people who have light chain amyloidosis, right, do not live as long. There's very little in the way of treatments.

And as they progress through their illness, the reason that they die earlier is that the light chain amyloids penetrate into all their organs such that the organs shut down because they become gummed up with this, amyloidogenic peptide. K? Mhmm. Now if we look kind of explains a number of things that people might be seeing out there in the world. Yes.

So this is this is something that I would encourage people to maintain, which is that because of the nature of this type of disorder, it'll it can manifest in a number of different ways. Okay? Cancer is one. Neurodegenerative is another. In this instance, if your circulatory system is filling up with this undigestible amyloidogenic peptide, you're gonna develop all manner of cardiovascular and thrombotic type tissues or or or diseases.

And, again, I you know, if there's no desire on behalf of the authorities to look at this, because it essentially, the blowback would go to them anyway. The buck stops with them in terms of how the public thinks. Right? And if people have lost loved ones, either due to exposure to this synthetic virus or due to a new just to, force public health measures using novel techniques that have never been deployed on mass before, where you where you've made the body express this toxic peptide that can act as a seed, right, to catalyze something into the well, I should be wearing gloves. Where you're getting grams of material post exposure.

That's pretty dark. It's very dark. And all all of it fits under the, the umbrella of proteinopathy. Proteinopathy means misfolded peptides, and there are hundreds of identified peptides now that are implicated in in any number of diseases. Now I would just encourage people to think about what what would they be looking for in terms of a battlefield incapacitation agent.

You don't want you can't wait for the opposing side to develop tumors. You want something that, initiates a quick knockdown of their fighting effectiveness. And in this instance, if you can gum their blood up with a nondigestible clotting agent. And clotting agents in and of themselves are a biowarfare. They're a chemical clotting agents, right, that they can deploy.

I mean, this has, sort of implications for well, I guess, we haven't tested yet, but whether these, seeds, if you wanna call them, are present in saliva, blood. But that's something that's something we'll get to. Okay? Yeah. So we we haven't finished yet.

We've just started the analysis into into what we're looking at. Okay? So okay. We can we can start looking at the limits of, the light microscope using UV. And, hang on.

Let me just check that's not the wife. Alright. Let me just do this. Right. So you you're just looking at higher magnification here, negative versus positive stained tissue, and you're just getting a closer look at the sort of punctate lesions, which I would hazard a guess.

So not not hazard it's not guesswork. Hypothesize more aggregations of beta stacking. Okay? And can we answer that question? Is there is there a way to determine if there's beta stacking involved in this structure?

Okay? So, what we can do is, you can then increase the magnification that you use. So you go beyond light microscopy. This is the scanning electron microscope we have. And now we're gonna go down in where you're going in tens of magnification previously, we can go in thousands of magnification, power of magnification.

K? And so what are you looking at here? So using the microtome that you saw previously, I'm cutting at its at its limit, basically, which is approximately five micrometers. There's gonna be some jitter in that just because it's at the limit. But I've taken the thinnest cup possible that I can of the tissue and then tried and it's very hard to get that off and laid out in as, even a form as possible just be just just because it's so thin.

Okay? But and and so it has, a somewhat sort of folded appearance on the bottom half of the image that you're looking here. Above that is just the the glass slide itself. Okay? And this is at 25 times.

So this is in the range of a normal light microscope. Now we're going down to 250 times, and you're going past what a light microscope can reliably do. And what do we see? Well, we see what looks like, again, sort of fibrillae type, structures that we saw at the light microscope level. And so like, the way to think of it is essentially sort of fractal.

Right? As you as you go down through the scales, okay, of or or power of magnification. Right? And so you'll see a sort of gross morphology. And then as you sort of see coherent structure, when you get to where you see coherent structure, which was the fiber matting and then what looked like, punctate, aggregations.

As we increase the power, you as above, so below, you're gonna you see the same sort of structure still there. And at this level, basically, what you're this power of magnification, basically, you're zooming in to, like, one fiber length or or cross section. Right? So light microscope is showing you a mat of fibers. Electron microscopy is now showing you what one fiber looks like in that mat of fibers.

So that's 250 times. This is a thousand times. So, again, what do we see? It looks like a crisscross network of, peptide, but there's also sort of bulbous aggregations. Okay?

And this will become important as we go through the next slide steps. Okay? And, yeah, again, just to reinforce to people that you're what you're looking at here is at the what was a light microscope level, one fiber. Right? So I just wanna reinforce this point.

Right? So if if I zoom in, you're basically looking at one fiber. I don't know if you can if my cursor is on the screen or not. Right? So you're basically looking at one fiber here.

Right? Go down here. Zoom. It's amazing what the machines can show you. Yeah?

Yeah. Yeah. And the There's no there doesn't look like any nanotechnology there, Kevin. No. That's this is the and it's been incredibly frustrating because, you know, I've had a sort of public role in, you you know, not just pushing it back against the orthodox and the corporate quangos, etcetera, but fighting against the the, the stupidity that sort of emerges on the I don't wanna say stupidity because it's that's not fair to some people who have concerns around things like synthetic biology.

Synthetic biology is is a train that's barreling down the tracks towards us just as a consequence of the development of technology across multiple domains. Okay? I'm not I don't think that you're seeing that in this instance. You're seeing something else. What you're what you're seeing, again, just to reiterate to people, is the deployment of amyloids across a well, they've turned the planet the whole planet became, a battlefield in this instance.

And, it it whether you could perhaps argue that this was a test for them. Right? Do do these do these make good weapons? Right? When you when you deploy them, can you can you knock out 30% of a fighting force if they don't know it's been deployed and they will develop blood clotting, you know, sort of instant blood clotting such that their oxygen carrying capability is reduced by 50%.

Right? So, like I say, you see this crisscross mesh of, proteinaceous fibers. Now, you know, you could you look at most structures under electron microscope, and you'll see you'll see something similar to this. And that, you could say, is, the normal, the normal features that you would expect to see. But in this instance, when we go down now right?

So this is a a thousand times. K? But when you go down to 5,000 times, what you see here and it's it's only because I'm familiar with looking at these structures that this was standout abnormal to me. Right? There are very clear nodular forms on these peptides.

K? And if you can see that. Right? So where my cursor is here, here, right, they they sort of stick out. And if we look at this primary primary branch here, what you see is fibrin should be a long, smooth, rope like peptide that essentially just overlays itself to form the network around which platelets and, other the other tissues that form a clot, aggregate.

Okay? Here, what you're looking at is a and so you're looking at very abnormal structural properties that stand out to someone who's familiar with looking at peptides in and of themselves. Okay? And those are there's these nodular forms and also what I would point out to people is that pay attention to this, thicker filament. Right?

And, in biology, often what you see and so the best example that I can give for people to think about is often when you look at a tree, you a tree, as you look at the trunk, has a sort of twisting effect to it. Right? It sort of starts at the roots, and it sort of has a rotation to it as it goes up to form the branch branch area of the tree. But the trunk often, if you pay attention to it, you'll see has a a rotation to it. And generally, in biology, I wanna say it's a right hand rotation.

It's the right hand rule of thumb, and you can get into all sort of metaphysics around, electrodynamics and, what that how that relates to the body. But in this instance, what you're seeing is is you're seeing a faster twist, so rotations per unit of distance than you would expect to see in normal healthy tissue. So imagine taking a piece of, if I have a piece of string available, but, I don't. But if it so if you took, like, a normal piece of rope and then you started to twist both ends, what happens to that, rope? It starts to tighten.

Right? And then it it it'll sort of it it just it starts to deform. Right? Just just think this simple thought experiment. Take a shoelace, start twisting it at either end, and eventually, it sort of shortens, and the the normal turns that you would get get compressed per unit of distance that you would use to measure.

Right? And that is one of the primary features that we use to describe in the scientific domain whether a peptide is normally folded or abnormally folded. K? That make sense? And, again, I'm throwing a lot at you because So, basically, you're saying here, yes, what you're saying is it's it's proteinaceous that it is is that what you're saying that it it just proteins.

Right. It's just proteins. Folded yeah. Misfolded protein. Is that what you're saying?

Nothing else in there. It's just a big slab of misfolded peptide that, is inherently obvious if you have the tools to probe it. The tools well, the analysis that had been done prior to this, whether it's people looking at it and seeing there there was no analysis done. There was some mass spec done. And, unfortunately, the initial mass spec was done by, Mike Adams.

And, you know, his just reputation is, toilet paper on the Internet with respect to doing anything, scientifically consistent. Okay? And, again, what he does is he's trying to drum up audience numbers, and so we'll immediately go to the more outrageous, descriptions. And he was talking about it having, circuit like properties. No.

You're looking at just misfolded peptide, just lots of it. Yeah. And the reason there's lots of it is because of the peptide that's involved, which is fibrinogen to fibrin, which your which your circulatory system is full of, in preparation, should you slice off a finger using your, chainsaw of a weekend. Right? Right.

It's just it's just the nature of the, You've got your body's got so many ingredients to make this if it misfolds. I mean, I think that was always a concern of mine. This whole nanotechnology idea was where is it coming into the body to make such a huge amount of whatever, you know, these Well, the reason it's such a huge amount is because you have so much fibrin fibrinogen Yeah. Per ml of blood. Okay?

It's like but in blood, you'll have red blood cells, platelets, and one of the primary, you have albumin. And then one of the other most common peptides are those there for clotting, furoinogen, which is there converted into fibrin. Okay? And that forms the clot. And the

reason you have a lot of it is sometimes, like I say, you can have a massive wound, and the body needs to pile loads of, loads of this peptide in to plug the gap.

Otherwise, you bleed out. Right? And so there's there's nothing there's nothing unusual here. I'm not that's not that's the wrong word. There's nothing spooky or mysterious here.

It's the fact that there's a misfolding of fibrin to fibrinogen sorry. Fibrinogen to fibrin, the clotting peptide that that is causing this, mass to appear in individuals. That's what that's what I would posit and hypothesize. I haven't been able to witness firsthand the extraction of these clots, but I would I will I'm trying to make sure that that whole chain of, investigation and necessary, legal steps is taken care of so that we can we we can push forward. All we can do is just, you know, one of the few wise things that my PhD supervisor said to me is just always put your best foot forward in this instance, especially when you're dealing with what is essentially an unknown that exists at the edge of, common understanding, which is the whole point of doing scientific investigation.

Right? So there we have in I'm gonna see in this instance, black and white, prima facie evidence visual evidence so you can see that it's misfolded. Okay? Now well, show us that it's misfolded. Let's me, why am I missing slides there?

Oh, I know. I I'm looking at the wrong presentation. Yes. Let's go here. So So is this what we would see under the scope in terms of other neurological diseases?

Was it something Well, you just don't have this this volume of material to look at. Oh, okay. Right. So even in something like Lewy body dementia, Parkinson's, etcetera, when you look at the tissue, when you've stained you use the same dyes that I've used here, you'll have little punctate spots that are essentially intracellular inclusions or often or in depending on the disease, will sometimes be extracellular, but, encased in the myelin or in the extracellular space. You know, the characteristics between different proteinopathies are varied, nuanced, and, essentially, you have to spend a lifetime studying them in order to have a good, image in your head of what they are.

Now I'm I'm confident with respect to Parkinsonian like neurodegeneration because I've spent twenty five years investigating it and working with it, and, I I have a extensive work just hands on experience with what that, tissue looks like. I can I can cross reference that to other disease states? But do I do I have the type of in-depth knowledge that I have with respect to Parkinsonian lightener degeneration? No. I don't.

I I can understand what person someone who has spent twenty five years working in Alzheimer's is saying because, you know, we've we've come to a common understanding that there is this amyloidogenic stacking of hydrophobic b depleted sheets that occur in proteinopathy. K? Now, in the summer of twenty twenty four, this manuscript came out, Fibrin

drives thrombo inflammation and neuropathology in COVID nineteen. Now this this was a very, very good paper. Very I encourage everyone, please, take a snapshot of that of what's on the screen here.

It's a open access manuscript. And I think it's I don't think it's on the screen at the moment. It's not sharing? Not that one. Oh, okay.

The primary plots. Yeah. Yep. You know, we were looking at this slide. Okay?

And, so I was saying that there was a manuscript published. This was from University of California, San Francisco twenty twenty four, and title being fibrin drives thrombo inflammation and neuropathology in COVID nineteen. Now what it and and this is gonna present a degree of I don't wanna see. Well yeah. Are we are we brutal?

It's gonna be degree of cognitive dissonance here as people who sit and have their own view as to what what's caused what, are gonna get challenged by the just extent data. And like I say I said earlier, my job is to remain as objective as possible in the scientific investigation such that I'm not led astray by my own confirmation biases. K? And what this manuscript has shown is that and very nicely. Oh.

Right. So that's behind that is the image that we were looking at before if I sort of zoom in. Right? So here you can see an example of what on the left hand side, human plasma, fibrin, and, you can see, like I said, it has this smooth rope like appearance. If you expose it to the spike protein, what happens?

Oh, it starts developing abnormal twists and little bumps and nodules that we were looking at on the, image that sort of in the frame behind. Right? And we were looking at on the previous slide. And so they're looking at a protein up proteinopathic form of fibrin in in controlled experiments. And, look, I've I did my PhD primarily at UCSF.

Okay? Very, very high standards of work come out of there. A lot of other deranged stuff does as well. But, you know, in terms of the the validity of this research, I'm fairly confident that they would have gone through all the steps required to, ensure that it's up to the highest scientific standards available. And I know I know know people have just a distrust of that, and you you're sort of you need honest brokers and scientists to be able to tell you, okay.

In this instance, this looks legit, and this says nothing about the source of, COVID, etcetera. It's just looking at the pathology that we can see when there's spike protein exposure in this instance. So for all the people who think that long COVID is just, vaccine injury, that's very obviously not the case because there was a whole cohort of individuals that got impacted by, the long term sequelae of COVID infection prior to them even deploying vaccines. There are I I know many people who have, been, yeah, just impacted severely post infection, and they made a point of avoiding vaccines. That's not to say that I don't know.

I know many, many people as well who have been vaccine injured, but, basically, peep people have sort of got it into their heads. It can only be one or the other. Whereas, you know, the world is a more complex place than that and more nuanced. And so you you you're required to sort of you know, actually, you gotta take the evidence that's out there and, you can't just eject something because it doesn't fit your, your current mode of thinking. Okay?

Now what we can say is spike protein in this experiment is giving us this misfolded nodular like form of prion protein. K? Now it's not a leap of the imagination to say that, well, if you go and gene transfect people and we have long term expression of spike protein because of the nature of the way that they made those, products. Right? Methyl pseudouridine to prevent breakdown, and they've already come out and admitted that because of methylpseudouridine, you get a frame shift that can occur and basically you get a noisy expression suite of peptides that get released.

And if the and so why why do you see that nodular bumpy surface? I would argue vociferously that there is a epitope signature that's part of the spike protein, which has amyloidogenic catalytic effects aimed at the prion protein. Now doctor Steven Quay in, I should I I should see if I can find that. But, he put out a preprint looking at the, n terminal domain of the spike protein. This is important point, actually, that we should we should bring up.

Give us a moment. Yeah. It's this one. Now it's sharing application, right, rather than if I drag this over, does that you see that? Not yet.

No. Just keep going. Yeah. It's it's, I have to go through the whole thing of, sharing screen because I keep sharing application. Let's do this.

Stop share. Let's share. That one. Leave it. That one.

Yes. Sure. Alright. So, Doctor. Stephen Quay is, an end of I've I've never I know people who know him well, but I've I've never had the chance to really speak to him personally.

But, what people have said about him and the work that I've seen him produce, I I'm of I I don't have concerns about his scientific motivations. Okay? So this was this was a, manuscript that he submitted, put put up onto a preprint server, but I think is of critical importance. So the thrombo inflammation and neuropathology sequence motifs of SARS CoV-two spike protein appear to have been engineered into the virus. And so if something's been engineered into the virus, it's also being copied into the vaccine itself because, basically, they've gone for a codon optimized copy of what the spike protein is, turned it into a synthetic mRNA molecule, and then put it into the population at large.

Okay? And basically bypassed immune barriers. So a landmark paper, and this is the paper that I just showed you, fibrin drives thrombo inflammation and neuropathology in COVID nineteen was published in August 2024 that concluded the mechanism of the thrombotic and

neurological symptoms following a SARS CoV-two infection, often called long COVID, is attributed to the binding of fibrin to discrete proteins of the spike protein, specifically free and terminal domains. This paper is a high impact publication with greater than a 10,000 views, placing in the ninety ninth percentile. Here, I examine the regions of the spike protein that bind to fibrin, fibrinogen, or both.

The n terminus of the Sprite protein contains the three strongest binding peptides, and surprisingly, these regions are also the free insertions in the protein sequence that are unique to SARS CoV-two and not found in natural sarbicoviruses. So if people have the stomach for it and the patience for it, if you go back to the beginning of the we shouldn't call it a pandemic. What we were talking about is a, mass casualty event because of biowarfare programs. Yeah. They are.

Important. Yeah. Yeah. Because if you call it a pandemic, it just it it disarms the cognitive apparatus as to think of it as something natural. Okay?

It's not It's like we shouldn't be calling them vaccination injuries. Yeah. Right. Yeah. It's it's yeah.

People just need to be careful in this domain. I I encourage you to I I know it's easy. I do it. It's a it's a lazy thinking, but it's not. It's a it's a biowarfare related mass casualty event, okay, that may rather than being accidental, by any accounts, could have been, initiated, and it's a binary more than a binary, attack because you not only do you have to have the virus itself, you have to have the secondary component, which is the gene transfection of the, the toxic peptide.

But you also have to line up all those institutions, remember, to get them to work in lockstep so you get as many people as possible so you can hit 5,000,000,000 people with whatever, 13,000,000,000 doses of, vaccine or or gene transfection. Okay? And so that's that's what we're doing. So it's it's important to think that. Right?

So in in this instance, we there was suspicion right from the very start because you could take the sequence that was published, go and look on BLAST, and see where the, what's how this peptide with associated with this pathogen, how it lines up with what we know about what we've aggregated from many, many, studies that are that are looking at these peptides, which Wuhan Institute was one of those organizations doing this. And there are there are nucleotide sequences that when you do that nucleotide by nucleotide sequence comparison, are standout because they're not in there. And in the case of SARS CoV two, there were a whole bunch that had homology to HIV. Not only did they have homology to HIV, they were in very specific regions of the spike protein such that it made them highly functional rather than just being a sort of, coincidence that they were and they're just buried in a, substructure of the peptide that the they're not really part of the pathology to do with the, the virus in and of

itself. Now, doctor Quay goes on to say, all pre pandemic sarbecoviruses have either a partial deletion in these regions or have protein amino acid substitutions that are non conserved and therefore would not support fibrin binding.

In addition, the free inserts also correspond to regions of the spike protein that have been shown previously to have high sequence homology with the HIV GP one twenty protein. GP one twenty is an HIV surface protein that binds to a host cell surface receptor, CD four and t cells, and facilitates cell entry to begin infections. In comparing the immunological and clinical presentations of HIV in COVID nineteen patients, the commonality of d dimer production, CD four plus lymphopenia, neurotropism, and interleukin 10 expression strongly suggest that these sequence homologies are clinically relevant. A conclusion that the path sorry, pathophysiology of long COVID is based on the insertion of spike protein motifs with secret numerology that mimic HIV g p one twenty protein motif properties and that these SARS CoV-two motifs are not found in salbacovirus subgenus strongly suggests that these inserts were design features in the synthetic assembly of SARS CoV two, meaning it was a deliberate insert into the biowarfare agent to to initiate a specific effect, the targeting of fibrin such that it begins to misfold and you develop the sequelae of long COVID, which is characterized by extreme fatigue, neuropathy, and, you know, what's what's essentially a chronic, a chronic lack of oxygen perfusion of deep tissue.

K? Let's see if I can get back to PowerPoint. So, here here so from these calamari clocks, okay, you can just reject all the nonsense that has been said about them. And using the correct tools, you can look you can look at it and see that it's a misfolded peptide that lines up with and has a causal mechanism related to what's called the n terminal domain of the spike protein in and of itself. Right?

So contact between one and the other will cause this misfolding. And because of the nature of fibrin and just the the amount of it in the body, it's gonna cascade, and the spike protein could be long gone. Yeah. So, I mean, I I know we can't possibly answer it based on what we know about this because it's still kind of, I would say, experimental. Yeah.

We we're sort of going into the great unknown here. But what is the prognosis? Do you think like, what what sort of timelines are we looking before we're seeing a lot more injuries from from this? Well, as a as a endemic pathogen, it still does the rounds. My family just got a a big dose of it after Christmas.

And, you know, we had we had clinically identified influenza, prior to that hit hit all the kids, etcetera. And a few months later, COVID. And, yeah, they were they were distinct illnesses in and of themselves. So for those that wanna say it's just flu, they can they can I'll keep my language. Well, We can hope we can build we can move beyond that.

I think there's enough evidence to say that all of our It's overwhelming if you want to look at the evidence. Right? I'm not even finished yet. There's more. Right?

So you you asked about the elemental analysis. Right? And so this just this is a technique. So when you're using scanning electron microscopy, you can do something called EDX mapping, right, which is a, electron dispersive x-ray mapping. So you're hitting it with a electron beam, and you can get a sort of form of, mass spec, and it'll tell you what that, structure is is made of.

And what do we see? Calcium, oxygen. Silicon is the glass slide on which it's in. Chloride and sodium is the, buffer in which the peptide was, kept and processed. Magnesium, aluminum, aluminum possibly because the stage on which the slide is sitting, is made of aluminum.

Calcium, phosphorus, sulfur, and nitrogen. Now do those ratios seem abnormal? I don't know. Is it the I will do that ICP mass spec myself, and we'll get a measure. And I'll get a measure across many, many more individuals than is currently being done.

And we'll see. And I'll I'll see if I can, sneak some regular clots in there and see how they were abnormal. You would think, let's say, theoretically, the phosphorus was was high. Wouldn't the body have to, you know, find that, like wouldn't it be causing you know, where does that grab it from in the body in terms of balance and home Well, scavenge it from anywhere. Right?

That's So it's not like you would, say the food intake is gonna make any difference whatsoever for people. Well, this this is a good question because we don't know if there's some environmental factor that may be another trigger in the formation of the the macro formation of this phenomenon. I I can't exclude that at the moment. And People are eating high phosphorus foods, you know, like, I know we haven't I don't I don't know if I don't know if it would be that in and of itself, but, the, oh oh, I'm just gonna just just say I don't know at this point. Right?

So we were looking at we could be have multiple triggers because we don't know why say some people who are exposed to it within a certain family react differently. And that comes down just to the individual's, own genomic makeup, environmental makeup, their health status, etcetera, etcetera. There's innumerable factors And the prob one of the issues that we just or I find all the time is that people will have a binary way of thinking about any problem, which is, if if that happened, then so if they got COVID, then they should get x y z when we know that, no, that's not the case. Some people will develop all the all the problems, and some people just get it, and it's it's not an issue. Right?

And Or could be a delayed reaction perhaps? Or or delayed reactions, and we just there's the clinical picture is so large. And, again, it's gonna come back down to the, honesty and veracity that we can put into the institutions, and the the clinical frontline as to what they're seeing. Now right now, there may be discussions going on between, you you know, research hospitals that are saying, hey. You know what?

We are seeing a whole bunch more clocks. Perhaps we should be looking into this. Perhaps, but for we know, there's probably a big multicenter grant and, proposal submitted saying we need $50,000,000 over ten years to look at and understand this phenomenon. Perhaps they are looking at it. I don't know.

We don't know. Right? That you're just always gonna be working with a whole whole degree of unknowns as we, as we the public has to try to sort of, work in real time with the events happening as they as they do. And what are we and why are we what are what are those events? It's people dying unexpectedly.

There was a study out in JAMA last week. So it might have been the week before. But they're just admitting, oh, cardiac events in marathons have shot up, whatever, you know, significant percentage since 2020. And particularly, I wanna say 2023 was a sort of standout year. And that's probably we don't know the data for 2024 and 2025 yet because we don't know if they've they're still probably doing the analysis.

K? But the the the data is that dire enough that people who work in these places, understand that it affects all of us. And Mhmm. Even I know the ABS is no longer publishing excess deaths, the actual numbers. They're doing it as an estimate.

So a lot of this data that we don't get to see, no one's getting to see it. Right. The UK How can we make how can we find the sign science of what's actually going on if we don't have the data? Right. And The UK so, if I shoot up my slides.

So I was there was, what was he called? See if I can find it. There was a guy on Twitter who was doing UK, deaths. Right? This is it.

What was he called? Outside Alan on Twitter. And the you know, what are you what are we looking at here? You're looking at all cause mortality. So this is weeks ending March 27, October '23.

Okay? And, where it's red and black, you're seeing extreme increases in all cause mortality. Now the disturbing thing to look at is that as we go so as we you work down, you're working across the years of the mass casualty biowarfare event. And we see and if you look here, so this is the age age categories across the top. Zero to 24, 20 five to 49, 50 and older and older you get, right, is the, as you work left to right.

Now at the outbreak or the start, it generally hit the older population and generally sort of stayed like that. You know? You're still seeing some in the first year going into 2020, '20 '20 '1. Right? So this is not vaccine related.

Now some will argue that this is entirely hospital protocols. Maybe. I'm I'm not so I wouldn't speak so confidently as to the source of these deaths. Right? Especially especially when it's happening, in the young.

And you you are dealing with a novel pathogen that we do know has this coagulopathy component to it. Now for sure, were they taking people because their blood oxygen saturations were dropping so precipitously, and were they putting them on ventilation? Yeah. They were. But that doesn't mean they would have got out of the disease state that they were in had they had they not had vent, ventilation done to them.

K? As you proceed through the years, high all cause mortality so on the right hand here, as we look at the latest data that we had, available to us, But it goes back to normal levels. Where you keep seeing excess death is in the younger cohorts, the 25 to sort of 50 year old cohorts. And that's that's your working population. That's your parenting population.

That's that's that's an assault on the carrying capacity of, of any country and society. And, you know, you you can argue the till the cows come home, what's the source of that. I'm not gonna do that right now. All I'm gonna say is I know that there's a because I'm looking at it. Right?

With this instrumentation, I'm looking at misfolded fibrin. There's the cause for the, what I would say accounts for the majority of the disease burden that we're seeing. K? And how that how that maps out, etcetera, is complexing, and it's not the topic of discussion for today. K?

So Where did I That is a very interesting thing. I actually haven't seen that that before as to why. So the older population isn't matching that black red. Mhmm. Yeah.

It's and it it's disturbing. And what did The UK do in I wanna say at at the turnover of '23 and '24? They basically changed their whole formulation for reporting on deaths. And so you can't do that when there's a obvious signal in your in your data that, that and it you could perhaps understand it if it was the elder end of the population. But when you're seeing such heavy, impact in the younger generation, and you you don't go changing how you record and report deaths to the public, but they went ahead and did it.

And that means, you know, under the current circumstances, I'm inclined to be less, confident in the, the output of the work that they're doing. K? Now I don't know. Oh, well, they're not the only ones. You know?

So thanks to the they passed that message around, that memo. Yep. So that's not the one. This is an older presentation. So, let's go.

This one happened. I've got so many presentations open that I'm trying to find Brahman spectroscopy. What I make a sec. Just because I I I think this is, important. It's okay.

We can edit it out if it takes Right. A lot of time to find it. Okay. So using, I'm annoyed because well, okay. So, actually, this this slide if I just copy this.

So I've I've had to expend a lot of effort into, you you want to be able to categorize the, the molecular structures that you're seeing. And in order to do that, the one of the more cutting edge ways to do that is through something called Raman spectroscopy. K? Now, what you're looking at see this little box here? I'm moving around.

Yep. Right? So as as we're beginning to learn about, the the fundamental nature and structure of amyloids, we can begin to say, what do they have in common across different protein proteinaceous species? Okay. And what do we have here?

So we have alpha synuclein at the bottom in light blue, amyloid beta, apolipoprotein, RPT, I'm not sure about, and I would have to go and read the manuscript. But the key takeaway here is is that at around 1,700 centimeters, which is just a essentially, you can just think of it as a arbitrary it's not arbitrary words. It's a measurement of, vibrational activity under something called Raman spectroscopy, where there is a increase in signal relative to noise in the background. And, as particular, wavelength, you can say this corresponds to this structure if you were looking through other more canonical methods like, western blotting or other mass spec type analyses. In fact, mass spec analyses are not as good at looking for this, amyloid stacking as, as the, Raman spectroscopy is.

Raman spectroscopy is, in fact, turning out to be one of the best methods that we have. Problem is is that a Raman scope will cost you a million dollars, and very, very few people have access to it. I'm lucky I have access to it. It's because of that scope, I was able to reject, the nonsense coming from, the graphene nanobot, side of the Internet. And what do I see when I take the slide?

Literally, the slides that we were looking at earlier, right, which which I've just shown you through the light microscopy, k, through down through the electron microscopy. If I do Raman spectroscopy, what you can see is in this is you get a peak around 1,700 centimeters, which says, yes. There's an amyloidogenic core in that in that peptide that we're looking at. K? So now we've ticked off another one of those boxes, and it's one of those boxes which is at the bleeding edge of diagnostics with respect to understanding the molecular properties of, any protein that you wish to be looking at.

Alright? Now, that then brings us to, this, which is RT Quick. So RT Quick stands for, real time quaking induced conversion. Again, I explained earlier in the, interview. You take a well plate Something something like this.

It's got 96 little wells in it. You see that? Yep. Right? And you fill them up with different combinations of peptides and whatnot.

And the idea with RT Quick is that, you take and it's primarily used for looking for Creutzfeldt Jakob, chronic wasting disease. You're seeing an acceptance for, I wanna say it's not is it alpha sign nuclear? Well, all all the sort of peptides that you could generally get from a spinal

tap that could that could point to the the there being the onset of neurodegeneration even though it's not, it's not visible, the person is still in the what's called prodromal phase. Okay? You might see hints of it, but the full disease expression isn't there yet.

And you can take, you can do a puncture of the spine. Takes take the c CSF, run it through, RT Quick, and then see if you can cause the reaction where you take a seed amount of the peptide and then you run it against the, the so you take your sample from CSF. You have a normal bulk solution of the peptide and you see, does it does it cause the amalgamation into beta pleated sheets such that it causes an increase of fluorescence per cycle that you run. And this runs for, you know, the you can do anything from, like, twenty four hours to a week into in trying to sort of investigate this phenomenon. And, I've spent, like, the last year essentially trying to use recombinant peptides just to see if that prion signal is there that was reported by Hammersch Sturm and Nystrom.

And I can point to that work here, hopefully. Get this. Right? That's Hammersch Sturm and Nystrom's work. And just this little cluster down here, spike five thirty two seed, specifically causes the aggregation of prion protein into the scrapy form.

I can say I was unable to really replicate that. Some signals, but not really very strong. When I did, when I took a seed amount of, the misfolded fibrin so this is me running, like, all those little squares. That's basically a representation of all the, wells that I've got running under reaction for the RT Quick. And when I was doing this, the, I was like, you know, I'll just test quickly, the clot against plasma.

So I, you know, I draw blood. I separate out the plasma. I put it into a well. And after a year of using or or trying to get a signal, I get a massive signal with RT Quick with this seed amount of calamari versus my plasma. Right?

Which is what these two boxes or two bars here are indicating. Everything else, all the reactions I'm running, there's whole combinations of different peptides or common peptides running, and there's nothing happening. It's all within sort of, control range. The two wells in which I had enough plasma and, yeah, enough plasma to fill those wells and then put in, a small seed amount of the calamari clock. Yeah.

I got I got a huge signal. Were you surprised? Was I surprised? After, you know, a year of trying with spy? Well, yeah, it's it's it's it was nice well, let's say, when you're building out a lab, especially when you're sort of starting from scratch and you're working under the conditions that I am, which is you are like, every penny counts because you've gotta be very strategic in how you deploy your resources.

You wanna be you you you wanna know that you're capable of capturing the phenomenon that you're looking for. Right? And, you know, after a year of trying with recombinant peptides and, you know, primarily around spike protein, prion protein, etcetera, etcetera. I wasn't

seeing anything. And, yeah, there there is this sort of anxiety that a scientist is gonna feel that he's you're messing up somewhere in your protocol such that you you're you're it's your fault rather than the principles behind the study that you're doing.

Okay? And so to see a signal there, I'm like, the I'm I'm not doing it wrong. Right? I'd I'm I'm concerned about the validity of trying to use recombinant peptide. I think another validity.

You you just need way more infrastructure and there's I think there's just way more steps you need to do to be sure that the peptide is functional when you receive it in its lyophilized form. K? And primarily, those peptides are used for assay. Right? So, you know, do you when you do like a Western blot, if you've got a sample so you take a sample from someone's spinal, fluid, You wanna look for what peptides are in there.

So you ask the recombinant peptide company, make me this peptide so you can map out its molecular weight with a dye. And then you see the the expression profile that you get from your patient, and you see does it match what you what you think might be the abnormal peptide in this instance. And I'm talking I'm hurling at you a whole bunch of complexities with respect to the the, how these systems work and interrelate to each other. But, I'm hoping that just people will go back, listen to this multiple times and try to absorb what it is that's being said because you need to learn this stuff. Right?

And you must get on top of it for what it means. So, to see this signal is not surprising, but it's it's it's not surprising considering what we know when we've looked at the scanning electron microscopy. We've looked at the ultraviolet histology with respect to using fire flavin and light microscopy. The signal that we've seen with Raman spectroscopy, this is just sort of like the final horrible euphemism, but in this case, the final nail in the coffin. Right?

And so that suddenly that signal then opens up a whole bunch of, potentialities that, I'm I'm concerned about. And so immediately right? This and I only got that data a few weeks ago. Right? And so I'm trying to like, is it so let's try and pass out what this means with respect to the public at large.

So first off, I'm fairly confident that most blood products post COVID are likely contaminated with probably seed amounts of misfolded fibrin. K? Now you know what? That that was a question I was gonna ask you because I was talking about it the other day, which was there was a case in New Zealand. Yeah.

And I'm pretty sure that, we've we've we've heard that, you know, peep people have died from, blood. So so the baby where they where the state stepped in, the right. There was a case where the parents were not wanting, blood transfusions from the sort of public supply of, Yeah. I I think in that particular case, because it was so high profile, I don't think the baby just passed away. It wasn't that case, but I I I from my memory of that, it was that that family had read of another case or had been notified that this had been happening.

You see, I so I'd like to clarify. Right? So if anyone has the reports or the tweets, if they haven't been nuked off the Internet. But I I'm pretty sure that, they did do the the surgical procedure, and there were post surgical complications of clotting. Right?

That's that's what I remember. Now did the baby pass away? I'm not sure. I don't think so. But, yes, there were problems if if I remember correctly.

But, you know, memory being what it is, you know, I'd like to I'd like to see it in sort of, black and white. But, anyway we do have a precedent for all this in the whole infected blood scandal, that's have in The UK anyway. So it's not, a stretch to imagine that if, you know, there are the seeded peptides or something if the blood is contaminated with anything, literally, then it's gonna cause issues if, they're not doing any screening for it. So, that's what what very dire, but, you know, we do have a precedent which went on for decades where they covered that up. And it's only recently in The UK where they've sort of admitted to it and and given some financial compensation in the in Australia, they're still not doing that.

Mhmm. Yeah. And that's that's the machine, and it just it always is. And the people expecting some sort of snap of the fingers and everything to ride itself at the moment. No.

This this is you're a long, long way away from that. And because of the scope of what it is that we're dealing with, the anyone in any position where they're gonna be held accountable is gonna do anything and everything to survive to the point where they can pull their pension and get out of Dodge. Right? So, you know, the concern here, right, is suddenly, there looks like there's an infective peptide if this sample that I've been running has been out of the body for three months. Even though it's been out of the body three months, once it comes into contact with fresh plasma, it's able to initiate the, the misfolding and aggregation into a amyloidogenic form such that it such that you're seeing an increase in fluorescence.

This is a big concern, and so and it's one of the sort of primary measures around how you would define scrapy prion. Right? So you can take scrapy prion and you can take femtogram amounts, and it can be infective years after. Right? So the operational principle around which they work with prions is there is no safe exposure dose.

Right? And, you know, when they do the experiments, you know, it's literally one there's in terms of dilution, you can basically say it's one molecule or or one piece of misfolded peptide is enough to initiate the chain reaction under experimental conditions, such that essentially it's it's a, lethal pathway in terms of experiment. K? Now how much that relates to everyday exposures? You know?

Not everyone died from BSE. Right? Now what what's the injury rate after that? I'm not sure we really know. You know, again, misfolding peptides manifest disease in many different ways, and there may be whole manner of fallout from, the mad cow outbreak that we just we just don't we just put it down to it being part of the, background level of of disease burden.

Mhmm. We'll never know if really will be. I mean No. No. I mean, there was there was a study done over many years where they were using antibodies, and this was this was before really these amplification methods were available.

And they were doing antibody staining, looking at people's appendix, and the I I can never remember if it was one in three hundred or one in three thousand. I'm gonna I'm gonna let me let me just because that's, that's an important number to know. Chat g p t. Right? Examining.

Index. What was the population penetrance of CJD. Let's do let let's just see what So sample, 8,318 appendices and estimated prevalence, three point seven per million. So one in two thousand people is carrying the scrapy prion. Just but and that's a course measurement trying to do it with a microscope and just seeing if you got a positive hit with antibody staining.

I would imagine if they took RT Quick to those tissues, they would probably see a much larger number. But in this instance, they've got a number. The government can say, oh, we've done the study. Boom. It gets put into the the, you know, the libraries, and they they've done their bit.

Right? And they just hope they don't get called out on it. So, with getting back to what we're looking at here. So oh, we're seeing something that looks infective. It's been out of the body.

And this this would fit the canonical description of, prion like disorders, and that's a huge concern. So I'm I've been working feverishly to try to see if I can, figure out what's been going on. And so, you know, I had to wait to get, this, which is basically just a ultrasonic, disintegrator of tissue and what have you, just just to be able to sort of break it down into a fine, fine form where, you know, I was generally having to do it sort of manually. And in this way, I can get a finer titration of the the amount going into per well of peptide. Even though I'm saying before when I when I had basically a course, non ultrasonicated preparation.

I'm unsure of the amount of seed material that's going in just because of the limitations of the methods available in the laboratory. So, in this instance, now you can see that there's, like, a uniform, distribution in the beaker. Right? And in this instance, this is I've basically just diluted it 50 times. Okay?

And if that was scrapey prion, that would still, that concentration light up all the tests. And in this instance so you're just looking at these sort of black black squares. Or or sorry, black lines. Each one is a well. This is a control material, and I'm not seeing a response.

Okay? And that's I hope is a good sign that there is it's, we're not looking at something as severe as, mass exposure to, scrapiprion. But I still think there should be concern about what is or what had infective properties months after, coming out of a cadaver. Right? Let alone

what's considered live blood and put into the blood supply for, distribution to hospitals for, you know, whatever treatment, whether it's emergency or, you know, people who've got cancer that need blood transfusions, etcetera, etcetera.

That's, you know But that's not diluted. So I'm I'm so what what I'm guessing you're saying is that, like, in terms of everyday exposure, you're thinking that it's it's, you know, to, say, surfaces or saliva or maybe, obviously. I mean, we can get Do do I do I think there's a a risk from shedding to this peptide? Yes. I do.

But is it in the is it at the level of concern where I would be talking where you're talking about chronic wasting disease, scrapy, BSE, etcetera, etcetera? I'm still I I can't give a definitive answer because I'm literally just working through the, the tests at the moment. But So that's what we're we're we're looking at in terms of in terms of getting you funded and continuing your funding, is to assess the disease risk from what you've recently discovered. Mhmm. Yeah.

And, all all I can do is just iterate through the peptides and the the well, there's just many there's many, many different factors that need to come together. And, I'm, yeah, I'm dependent. Again, like, I don't go to the big money players and ask them for money because that comes always with strings attached. Right? I I would rather do this and, ask to people.

You know, what I'm what I do at the moment is I basically say, I need this bit of equipment in the laboratory. Right? And, you know, you don't have to send me money. Send me equipment. Right?

Just so you're sure that I'm not, you know, I'm not running away with it or doing anything, untoward. I'm I'm working literally every day with this at this problem. Right? And I have been for essentially the last five years. I mean, I think the question people always commonly ask, and this is sort of part of the cognitive dissonance, is, well, how do they stay safe, or how do they heal from, say, something like long COVID, issues with shedding?

Okay. So, one of the things that I found I can do is the let's let's see. Let's go back, approve this. No. Maybe the other way.

So there are there are two issues that you're dealing with. One is, those who have been vaccine injured and those who have long term sequelae from the infection itself. Both exist, okay, and are often struggling with significant disease burden and are often being gaslit by medical professionals in that their symptoms are dismissed or they're told that they're psychosomatic. And what I've found is is that, you know, the if we understand this clotting disorder to be, a sort of universal key to understanding the symptoms, then it's possible to be able to test individuals and just give get an idea of their amyloid microclot burden. Okay?

Now there's no automated test to do this, and it's not on the tick box of standardized tests that your your GP or clinician has available to them. And nine times out of 10, most people go to a GP, and you get you get five minutes and pushed out the door as quickly as possible as they try to work through their, caseload for the day. Right? And you going in and asking for specialized tests that are only available in very, very few places, just often doesn't get you very far. Right?

But what we can do is we can look at so this is someone who, is vaccine injured from Moderna. Okay? And here, what you're looking at is a this is what I see all the time now. It's a microclot fibrilliform, and I tend to see that well, I had to classify them. Punctate, generally circular type microclots, more, how would you say?

Almost like sort of aggregations or sort of like tissue, if you like, sort of tore up tissue paper that was sort of stuck together. And then there then there is this fibrillae form that often has a sort of rotational, element to it, and we've spent a lot a lot of time talking about this fibrilliform or or this twisting of the peptide as being at the heart of the pathology that we're dealing with because that's the protein proteinopathy expressing itself, right, as a misfolded peptide. K? So you take that. You process it so you can check it with ultraviolet, and then I can I can discern whether those objects that you can see under light microscopy are there and, start ticking the criteria for amyloid like deposits, okay, or amyloid like inclusions in the blood?

And, this, you know, it's unfortunately, again, it's not it's not quantitative. It's not quantitative like RT Quick. Now, potentially, if someone was gonna fund me, I could try to do RT Quick off people's blood if they're, if they have vaccine injury or long COVID and see if it's triggering and you get an amplification out of it with respect to fibrin. That's that's an approach to take. But, in this instance, I will I can take people's blood.

I can process it histologically. I can look at it under a microscope, and I can tell you I can give you an assessment of how much amyloid you're carrying per, sample of blood. Okay? And, what I can tell you just of the library that I sort of built up is about getting over 50 specimens now. And it's approximately half and half people who think they're well, not people who think.

People who've experienced vaccine injury and, again, are being gaslit by their health professionals in that they're they can't have a vaccine. It doesn't do that. Right? Or it's people who have, had contact with the virus and have not recovered and are having a significant hard time with it, brain fog, fatigue, etcetera, and all all the other, symptoms that we've come to associate with these conditions. And under the microscope using these techniques, I can't tell that if I was blinded to what was the source of their injury, it's very difficult for me to ascertain which would be which.

The type of microclotting that you see is very, very similar. And so, so there's in in a sense, there's a degree of sort of, there's a degree of sort of hope here that, a lot of the battle is people just trying to get the medical system to recognize that they have a problem. And there

are, there are even medications that you could be put on. Right? There's there are, antifibrinetics that you can take should you have a problem with, you know, they think that clotting is one of your issues.

This you know, these pathologies existed before. It's just we've got another way of inducing them that's been introduced to the environment. K? And, you know, I know some people are, certainly with some justification now, suspicious just of the pharma medical complex. And, you know, there are there are multiple ways to skin a cat in this instance and try to get symptomatic relief.

And, you know, I like I say, I've I don't I can't risk my scientific objectivity by saying, go and use x product. I can tell you Yeah. Collaborative groups that I'm using, I'm working with, are seeing very, very good results through their approach. And, you know, if people want to, I mean, in theory, you could do this blood test, do whatever things that you're gonna do, whether that's that's anti febrile medicine or natural things, then send another blood test Yeah. To you.

Yep. And then compare what's Yep. Effectively working and and what's not working. Yep. And that's what I've been doing.

And, you know, that's that's going a long way to sort of keeping the lights on at the moment. It's just me me assessing people's, blood. Now, you know, as a it it's not a full objective scientific research project. Right? Because I'm the people who are sending me samples are people who literally have issues.

Right? So that's always gonna skew your sample cohort. And so it's it's difficult to say again what's what's the penetrance into the population. But I'm pretty certain at the moment that it's probably higher than they're letting on right now. And and, again, the problem because of the the people that write to me are saying, yeah.

I'm I I have this. I tried to go to the doctor, and I'm I'm basically just brushed off. Right? Or I'm I'm given, you know, the it's it's incredibly frustrating because what happens is is that people get labeled with a what's called functional neurological disorder, which basically means they don't know the cause and basically are trying to say to you, it's essentially psychosomatic. And, in this instance, with this data, you're able to push back against that diagnosis.

Right? It's not a it's not a psychiatric illness. There is a there isn't there can be an underlying pathology. And then if there's a test Right. The medical system, they love tests.

You You get a test done. You hand it to your doctor, and you ask them to explain it. Yeah. Yeah. And but they don't they don't offer this test.

Right? Yep. I can do it. Right? So, like I say, if you wanna help and you wanna keep this research going and you have a problem, you want me to look at it, I will.

And, you know, usually, the price to do that is a minimum $300. I I won't even charge you unless you want me to actually, give you a report back. Right? I'll I can look at it and, you know, if you just if you just wanna add to the library of samples that I have, I appreciate that. Right?

Because it helps me build a better picture clinically of what's going on. But, yeah, if, if you've been I almost said abused is the it's probably not the correct way of it's just you're looking at a system with limited resources, limited time, and they have to deal with hundreds of thousands of individuals on a yearly basis. They have to find a way to sort of categorize you and, give you a, give you some treatment. Right? It's just that in this instance, labeling people as having functional neurological disorder is, is the incorrect diagnosis when we potentially can look for pathological signatures and say, look.

There's something we need to be treating, and there are methods to at least try to mitigate amyloid burden, especially if it's in an easier to reach compartment of the body as the blood. Did you wanna, bring up your website, Synapsic Labs? Yep. Why why not? That's, always useful.

Yep. So you can go here, Synaptic Labs protocol on sending blood samples. If you go down through that, there's a little video, free, four minute video of me just showing what you need to do and what maybe what I will do is perhaps I'll let me just play this because then I can grab a drink, and then we can sit there and I can I can we can just sort of decompress for a little bit? And then if you've got any questions after this, and then we can wrap up. Is that is that okay?

Yeah. Sure. Sure. Okay. So and I I'm not gonna listen to I hate listening to my voice, but you hear that?

This is the suggested protocol Okay. For administering I'm gonna get a drink. A needle stick to get a blood sample to send for amyloid burden testing by UV microscopy. First, what are the items that you will need? I suggest alcohol, sterilizing alcohol, and swabs.

These items are given in the list associated with this, clip. And although there are differences slight differences here, with respect to methodology, I'll be using a syringe needle rather than a, needle stick device that is commonly used for diabetes. But the primary purpose is to show you how to take the blood sample, apply it to the slides, in preparation for posting, to Japan. So I have two micro slide two microscope slides that I have ready, to put the blood samples on. I'm going to sterilize the finger with, alcohol.

Never easy, but, we just need a few drops of blood. So here it goes. So that's the needle stick made. I'll withdraw. And, hopefully, if we're successful, you can see that we have got some blood.

Let's hope we can get enough. Yeah. If you just squeeze it. Now I'm going to apply to the microscope slide. One, two, see if we can get some more, three, four.

What I do suggest is try to, even out the samples that you've put. A method to do that is just use another glass slide and just spread the blood out so it's a thinner layer. And you wanna be aiming for around two two by two centimetres of coverage, each one of these sides. Slightly larger than I would have wanted. And that there is the would be ideal for analysis.

Let these dry, overnight in a dust free environment and follow the rest of the instructions included in this methodology. Thank you for all those that are contributing to this work. Has my voice stopped? Just so if below that is links to, where you can get so I'm just looking at Amazon.com. Here, this is, it it and this is the bottom range of cost for microscope slides.

And you also there's a link to I used a hypodermic needle just because that's what I had laying around, but, I would encourage people to just get the little lancets that people diabetics use. Again, $7 off, Amazon and, sterilizing swab alcohol. Everyone knows what that is. I would encourage people to look for so I have a link, these types of casings if you can. Right?

So you can you can basically get two microscope slides in there. Best to send to you because, I have had cases where they've broken one even with, protection and what have you, just to sort of increase chances of being able to, get a useful, sample from. And, with that, I can give you or the person a, a and, yeah, again, unfortunately, there's just there aren't automated ways to do it, and, there isn't a tick box that your GP can, tick for standard, blood tests, unfortunately. You get you you might try for a d dimer test, maybe. But I think, the the problem with that is is that you might have elevated d dimer after initial exposure as the as the clotting is sort of initiated generally just as just because you've got systemic coagulopathy from spike protein.

But once that sort of signal has died down, the body, because of the, it's not gonna signal that there's sort of, like, a clotting emergency with misfolded fibrin. Right? And that fibrin is just likely to just continue to aggregate and aggregate till we end up with those, proteinaceous rubber like clots that we were looking at earlier. If people wanna spend a bit extra for the lab, I like this brand. It's called, it's Japanese brand, Matsunami.

Very clean slides. The cleaner the slide, the better for me because there's just less confounding objects. But if people wanna link to where I get those slides, and it's approximately about $50 for that is hundred slides, maybe. Yeah. Hundred hundred slides.

And if people want, you can get a smaller vacutainer than this, five mil of, blood. You might have more problems sending that via the post. If you're just doing the glass slides, there's no problem. You can just you can send that in standard post and just put, dried blood sample microscope slide. That gets through no problem.

And, you know, I few times a week, I get a sample delivered. And I've got a backlog at the moment just because the scope I had was out of commission for a month or two. And, yeah, I I like I say, if you if you think you've been hurt either through the vaccines or from COVID, here's here's here's one way that you can get a, diagnostic, especially, like I say, the and I know because I've been through it years ago when, you know, I got her via postviral, syndrome. It was very debilitating for many years. I don't know.

I still think you I have to struggle with it these days. But, yeah, if and people could people can reach me through that page. The, the addresses or their, contacts. And, if you have questions prior to trying to make the sample, you can email me. I say, the, like, the Discord is a, it's a rowdy, bar type of environment.

Just think of it like that. But if you can put up with that or you can just ignore it and you wanna reach me, Discord is a good way to do that as well. Twitter, not so much just because there's so much spam DMs now as a consequence. So it's, it's difficult. And, basically, I'm just deleting DMs in Twitter just because it's it's just all looks like nonsense now.

So, yeah, I don't know if you if you had any further questions. Can you can you close that screen down, Kevin? I can. Yeah. Oh, look.

Thank you so much for all of this. I think we've covered a lot in in the time. Did you ever test your own I mean, it's just curious. Did you test your own blood in it? Yeah.

Have you found, No. Mine's mine's pretty clear, but then, you know, I have access to the the countermeasures that are being deployed, sort of clinical, testing elsewhere. Right? And, you know, the well, am I I can show you the product. Right?

And Yeah. Just You might as well. No one else. No one else is doing it. You're not making a buck from it.

No. I'm not selling it. Right? It's just got nothing nothing to right. I I don't get I'm curious because I if I had access to that machine, you'd be testing, you know, those people closest to you.

I'd let's say, I'm more than happy to put you in touch with the people that are making this, and you can speak to them about their data. And, but, well, the version I have, they sell it. It's called Medicinals. The one I have is just it's just labeled for a different market. It's called Malecusan in this instance.

And it's a very, very high grade mix of, active phytochemicals in this instance and, basically, combination therapies to, one, induce so sort of senolytics to take out infected cells that potentially sort of zombie like cells that are just spitting out peptides, binders so that the spike protein that is released is captured and excreted and targeted molecules to again, it sort of trigger sort of apoptotic pathways and deal with, circulating. So one of the targets is

primary blood monocytes of the immune system. So what they find is that there's a high burden high spike burden in people with, who are suffering sort of, sequela either from vaccination or, infection. And, they find that if they by targeting those, exosomes where, you know, they're they'll find, you know, in extreme cases, they can find a thousand picograms of spike protein on average per, monocyte. They can reduce that down to essentially negligible values, sort of in the tens of picograms.

And, in severe cases, the, the one of the clinical approaches that's being taken is to do, heparinized, apheresis, which is essentially, taking the blood, like, essentially, like you would if you were sort of donating blood. Yeah. But it the blood is circulated through, a whole bunch of filters. There are there are different types. So hepre hepreonized apheresis is harder to get.

There are other machines which are more available, less sophisticated, called the eboom machines, And they they're getting good results with those, but the gold standard is the sort of heparinized, apheresis. And that coupled with, you know, sort of multi month treatment with these products, they're recovering, many patients. I would say probably way way more than the orthodox health systems. I'm gonna say that. I don't I don't get any money from sending that.

Good news, really, because I think, what we've sort of established today is that if there is testing available, like, you can obviously rule out if it's amyloids or or not by getting your blood tested, which will steer you in a certain direction. And if you are taking any kind of treatment, you will be able to also assess ongoing by doing another test Mhmm. Gathering whether it's working. And, of course, for scientific research, all of us, we'll be able to see what's working and and what's not Yeah. Not working.

Yeah. And that, stuff out there right now is is just, like, take this on blind faith. And if you're feeling better, or even using medical tests aren't necessarily sufficient because they're not testing amyloid burden. Yeah. And so you might even pass a medical test, but it's not actually Yeah.

You're you're still feeling terrible. Right? You can't get you know, people can't get through a working day. And, again, the the end diagnostic that they'll give these people, funk FND, functional neurological disorder, and probably slap first you on some SSRIs and, you know, make balances and antipsychotics. That will be their approach, and that's it's when we have tools available to us that can prevent that sort of misdiagnosis, I would encourage people to sort of, at least at least take it and try it for a few months, and it we can I'm pretty confident we can tell the difference pre and post, treatment.

A lot of people well, I said a lot, but, you know, I have got people that have contacted me who are already on treatment that just said, can you please tell me how it looks? And, you know, that's, it it provides a sort of peace of mind for people and just not just peace of mind.

You want something that the nearly, I would say, 90% of the people that sent me, blood. When I've said when I've written back to them with their results and said, look. Here you go.

You've got this, this, this. I'll send you a full, report on all the images that I take, and often I'll send people just a video record myself working through the slide and just giving my commentary, send that back as well. And, that gives people, hope and something to go to their health practitioner with and say, this is where we need to be working. And with that, you can you can start working towards, trying to regain your health. Does it are are you gonna get back to normal?

I don't know. I'm I'm not saying that. I'm not a doctor in this instance, and I am I work strictly on the research side. And, you know, from let's say, I don't I'm not making anything from this. Don't sell it.

Don't do, there's no, product page on my websites. It's purely, if people wanna, contribute because they think this work's important, that's available. And if they wanna test and they wanna send, you know, couple of couple of slides with your email and, with your medical history, please. Just, just put in a, page printed page or handwritten page of, you know, the basics, age, past medical history, what you're going through right now, current treatments. And, yeah, I can you know?

And it's it's working pretty well. And, hopefully, that's the end goal because we may never know the actual, you know, weapon system in its chest. Well, I I think we have a pretty good idea now, actually. I'm I'm I'm confident now. Right?

Whereas before, you know, there was always that little bit of ambiguity because the you know, they they weren't admitting to it. Whereas literally, oh, yeah. They've just put their hands up now and just said, yeah. Okay. So, yeah, they were ill ill military.

It is fitting in a way. I mean, when you think about it, we'll be Well I mean, it's taking a long slog, but, you know, like, the the pendulum has definitely shifted to the bioweapons angle. Yeah. But but they've got they've got a way with it. They've got a way with it because the only pea the only people that left are those that have been impacted directly, whereas the, you know, the population where they haven't, you know, they haven't lost loved ones.

They had no adverse events. They've got they've got their lives to get on with. And so, you know, by whereas the whole world was keyed in at the beginning just because of the novelty factor and, yeah, there were people dying. Again, people are gonna argue about the source of, the deaths, but there's certainly a increased disease burden out there that can't be argued with. The they managed.

Right? They really managed to kick that can far enough down the road that, yeah, they you're not ever gonna see the mobs of pitchforks and torches raining down. So are are you still feeling like we're sitting on a ticking time bomb, or are you feeling like there's a lot more

hope now that since from this time last year? Good question. And, look.

I'm I'm I'm working as fast and as hard as I can to try to understand the impact of what that, misfolded fibrin is. And, you know, I what I can say is, oh, I've seen it be infective postmortem. I would suspect it to be infective via, potential exposure routes. The problem is, you know, primarily, you know, a lot of your stool contains broken down, fibrin. Right?

As, as just the liver doing its thing. And the so I think there there is an environmental seeding, and I would say it's with high probability that blood products used on a day to day basis are likely contaminated at this point. And what about, I know everyone. There's there's been lots of talk on social media. No sexual contact.

I I don't know. I I hope not, but there's still the disturbing trend in drops in birth that is ongoing in many countries. And the argument is, it's kind of heading that way anyway. But, I I I would say generally look at the with the if you're in a in a sort of settled relationship, family, etcetera, it doesn't matter. You're passing backwards and forwards anyway.

So, you know? But, you know, if you're dipping into the dating pool, you know, probably suggest, you know, discreetly ask about their medical history. I don't know. Just the usual usual usual, What a world we're living in today, isn't it? I mean, it's Yep.

It's it's quite crazy. Is there anything else you wanted to add? I think I pretty much exhausted. I think we've done a really good job today, of putting it all together. Yeah.

It was it was quite, it's quite long. I don't know. If anyone wants to watch it all from start to finish, and they will get the big the big picture, go back to the last video and get the kind of background around prions and so forth that all tie together. Is there anything you you wanted to add, Kevin? I'll just otherwise, I'll just close.

No. I think I think that's it, Jeanie. Just I just hope the message gets out there. And like I say, between this time and last year, like, I've managed to work out, at least, at least to give people some idea as to this, like the the more complex proteinopathies, like the let me worry about that right now. Right?

What we can see is is that there's a blood borne one. Right? There's one in the blood. It's a kind of low hanging fruit in terms of trying to address people's, symptomatic profiles. And, yeah, there's there's a way to at least get a handle on that, and I can point you to the people who are, making effective interventions at the moment.

That's correct. That's great to know. So in closing, Health Alliance Australia opposes harmful health measures, laws, and mandates provides educational content. Right now, I wanna acknowledge the work of professor Francis Boyle, who died recently, professor Francis Boyle, who drafted the Bioweapons Act. And in our interview that was censored, he called for all BSL three and BSL four labs to be closed.

He also said, we, humanity, are in fight for our lives. No truer words. Our other sensitive video was with Melissa Jolley Graves and her testimonial from healing from terminal diagnosis of neuroblastoma, blood cancer. Both these videos can be found on our channel on rumble and bit shoot along with other important videos. Please feel free free to share this interview and information widely.

Cut, copy, paste, distribute. It's important and heavily sensitive. Get it out there. When sharing, please do link back to doctor Kevin McCan's website, McCan Dojo, and his Synaptic Labs website, healthallianceaustralia.org, if you can. I'll be putting all the links plus a report, which includes the slides, important information, links, that we've discussed here today, and also Kevin's report, on that page.

So it's all in one place. You can visit healthallianceaustralia.org to join our mailing list, become a support or donate. Health Alliance Australia, myself, and Kevin, doctor Kevin McCann are also on LinkedIn. You can join us there as well. Thank you, doctor McKeven McCarner, for your time today, and thanks everyone for watching.

You're welcome, Ginny. Thank you.